FUNCTIONAL DIAGNOSIS

Atkinson

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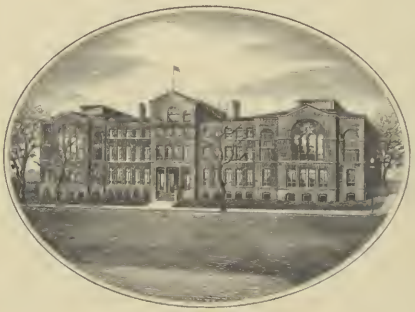

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Functional Diagnosis

BY

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BROMIDIA

Indications—Sleeplessness, nervousness, neuralgia, headache, convulsions, colic, irritability, delirium of fevers, acute alcoholism, mania, epilepsy, drug addictions, etc.

Dose—One-half to one fluid drachm in water or syrup every hour until sleep is produced. It is unadvisable to administer more than three fluid drachms in twenty-four hours except under medical supervision. Bromidia should be given to patients under conditions favorable to its action, *viz.*, in bed or reclining in a quiet, darkened room.

N. B.—Bromidia is supplied only in 4-oz. and 16-oz. bottles.

PAPINE

Indications—The same as opium or morphine, with less tendency to cause nausea, vomiting and constipation. Insomnia due to actual pain as distinguished from that due to irritability of the nervous system, which calls for Bromidia. A useful adjunct in cough mixtures. A safe opiate for children.

Dose—For adults, one fluid drachm pure. For children under one year, 2 to 10 minims. Papine should only be administered in accordance with medical directions.

N. B.—Papine is supplied only in 12-oz. bottles.

ECTHOL

Indications—In all breaking down conditions of the fluids, tissues and corpuscles, dyscrasias of the secretions, blood poisoning or tissue disintegration. In typhoid, eruptive fevers, smallpox, erysipelas, carbuncles, boils, gangrenous wounds, ulcers, abscesses, stings of insects, snake bite. Valuable as a local application in all pustular formations, fresh cuts and infected wounds.

Dose—Internal: A teaspoonful every 2, 3 or 4 hours, preferably after food.

External: Diluted with 2 to 10 parts of boiled water, or if necessary full strength.

N. B.—Ecthol is supplied only in 8-oz. bottles.

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Indications—Syphilis, hereditary or acquired, scrofulous and glandular ailments, gouty and rheumatic disorders, skin affections, chronic diseases of women, and whenever a safe, effective and reliable tonic alterative is required.

Dose—Two teaspoonfuls three times a day, before meals.

N. B.—Iodia is supplied only in 8-oz. bottles.

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RESPIRATION.

Retractions: The lungs may be considered as two large sacs which communicate with the external atmosphere by means of the trachea, the external walls of the sac being in adherent relation to the walls of the thorax. The thorax, outside of the lung-sac, is completely shut off from communication with the atmosphere and with the abdomen.

Owing to the adherence of the lung sac to the chest walls, a cicatrix in the former will pull in the external covering of the chest and produce a depression. These depressions are often seen in tubercular cases where there has been an old cavity that has cicatrized. They may be found in any of the intercostal spaces, but are most common in the clavicular and subclavicular spaces, due to apical cicatrices.

Pneumothorax: In cases of tuberculosis and other processes of lung degeneration, the lung sac frequently ruptures under exertion or cough, and air is forced into the thorax at each inspiration. This condition is known as pneumothorax. It may, of course, also result from a penetrating wound of the chest wall. (See Respiratory Sounds.)

Respiratory Sounds: The two types of sound heard in normal respiration are: (1) that produced by the passage of the air through the bronchi, called bronchial breathing, and (2) that produced by the inflation of the lung alveoli, called vesicular breathing.

Bronchial breathing sounds, as might be expected, are rough and sonorous, and are equally prominent in inspiration and expiration, except for the different force with which the air is driven through the tubes.

Vesicular breathing sounds are softer and more blowing in character; and inasmuch as they are caused by alveolar inflation, they are confined almost entirely to inspiration, the almost sudden recoil of the lungs in expiration putting a rapid end to the vesicular sounds.

Normal breathing sounds heard through the chest wall are made up of a combination of these two elements. The bronchi being at almost every point overlaid with lung (always containing more or less air), the bronchial sounds are not well conducted to the chest wall, and are therefore, in normal breathing, greatly subordinated to the vesicular sounds. This is especially the case in inspiration, when the alveoli are filling with air. In expiration, when the alveoli are recoiling and emptying, the bronchial sounds are becoming more dominant.

In infants and young children, in whom the lung tissue is relatively undeveloped, the bronchial sounds are normally much more dominant than in adults.

Bronchial Breathing: Anything which lessens the layer of air-containing lung between the bronchi and the chest wall, or increases the conductivity of the medium, exaggerates the bronchial element in the respiratory sounds and gives what is known as bronchial breathing. Chief among these influences, of course, is consolidation of lung tissue, which has both the effects enumerated above.

Bronchial breathing is therefore found in **pneumonia, tuberculosis, syphilis of the lung,** etc., over the areas consolidated by the process.

In **bronchitis,** the breathing is of a bronchial type in the early stages, because of the exaggerated roughness of the tubes; later, it becomes vesicular, owing to the emphysema and loss of elasticity of the lungs.

Vesicular Breathing is found in just the reverse premises to those which cause bronchial sounds, i. e., in those which increase the amount of air-containing lung between the bronchi and the chest wall. Such a type of breathing is heard in **emphysematous** conditions, especially **vicarious** and **surgical emphysema,** and **asthma.** Vesicular sounds are dimin-

ished without any special exaggeration of bronchial sounds, (1) by any condition which impairs lung elasticity, as **emphysema** in later stages, **oedema,** etc.; (2) by the presence of air or water in the surrounding thorax (hydro- or pneumo-thorax), owing to pressure exerted on the lung; (3) by pleuritic pains, limiting the movements of the chest; (4) by occlusion of air passages, lessening the entrance of air into the lungs.

Stridulous Breathing, a harsh whistling sound during inspiration, is frequently heard in cases of tracheal and laryngeal obstruction by **spasm, oedema, membranous croup, severe catarrhal inflammation,** etc., the sound being due to stenosis of the air passages.

Hiccough, as ordinarily seen, is merely a transitory spasm of the glottis, due to temporary irritation of the glottis itself or to some portion of the upper digestive or respiratory tract. Persistent hiccough, however, suggests some deeper lesion of the diaphragm, such as an ulcer or pus-sac.

Suppressed Breathing Sounds: Where the lungs are surrounded with air or water, as in pneumo- or hydro-thorax, both tubular and vesicular sounds are muffled, and in many cases **almost inaudible.**

PERCUSSION.

The principles of percussion are based upon anatomical rather than upon physiological premises. In general, percussion sounds may be said to depend upon the anatomical conditions corresponding to the physiological conditions accounting for auscultatory sounds. Generally speaking, in instances and locations where the bronchial sounds predominate, as in consolidations of pneumonia, tuberculosis, syphilis, gangrene, etc., the percussion note is dull or flat; where the vesicular sounds are in the ascendant, percussion gives a hyper-resonant note, as in emphysema, asthma, etc.

However, as the pitch and quality of the percussion note depend upon the relative resistance of the part percussed, rather than upon its conductivity, there are exceptions to this correspondence between auscultation and percussion. For example, a cavity in the lung will give bronchial sounds, because the vesicular tissue is gone from between the bronchi and the chest wall, but percussion over a cavity will yield hyper-resonance. Pneumothorax and hydrothorax alike diminish both vesicular and bronchial sounds, but the former gives a hyper-resonant note upon percussion, because of its elasticity, and the latter a flat note, because of its solid resistance.

INSPIRATION AND EXPIRATION.

Inspiration is accomplished by enlarging the capacity of the thorax by means of the muscles; the lungs, being adherent to the walls of the thorax, are expanded and air rushes in by way of the glottis and trachea to fill the vacuum thus created. Inspiration is, therefore, an active muscular process.

Expiration is accomplished by the elastic recoil of the lungs and the weight of the ribs—purely physical forces—and is a passive return to equilibrium.

Delayed Expiration: Any condition which interferes with the free expiration of air from the lungs exerts increased resistance to the recoil of the elastic tissues and to the gravity of the ribs, and hence prolongs the passive process of expiration. Bronchitis, asthma, nasal and laryngeal catarrh, enlarged tonsils and growths, all of which block the air passages, are prominent illustrations. The same result attends any pathological lesion which impairs the elasticity of the lungs. Delayed expiration, if long continued, eventually results in undue retention of air in the lungs (Emphysema, see below), and the external circumference of the thorax is enlarged (barrel chest).

Prolonged Inspiration is seen in any condition which interferes with the free passage of

air into the lungs through the glottis and trachea. As the same causes, however, usually interfere equally with expiration, the relative lengths of the functions is the same as those described under delayed expiration. Movable tumors of the larynx occasionally interfere with inspiration and not with expiration.

Forced Inspiration: Under any pathological condition which renders the performance of inspiration difficult, or which necessitates a greater number of inspirations in a given time, the ordinary muscles of inspiration have to be reinforced by the accessory muscles of respiration, namely, the sternocleidomastoid and those muscles which control the size of the nasal and tracheal openings. The involvement of the muscles in the act of inspiration will be found an unfailing indication of seriously embarrassed respiration, such as occurs in pneumonia, emphysema, pulmonary oedema, advanced tuberculosis, all those diseases of the heart and thorax which impede inspiration (hydro- and pneumothorax), and all conditions which obstruct the air passages, as laryngeal oedema, growths, membranous croup, laryngismus, etc. Those which ordinarily take part in the process are the costals, intercostals, scaleni and serrati.

Unequal Expansion of the two sides of the thorax may be due to an impairment of the

elasticity of the lung tissue on one side or to an immobility due to the binding down of the pleural sac by old adhesions (in which case the outer wall will be retracted as already described), or to effusions or tumors in the thorax (in which case the immobile side will be distended).

Litten's Phenomenon: In the position of respiratory rest, the diaphragm lies flat against the thorax from its attachment to about the sixth rib. Upon inspiration this muscular layer "peels off," as Cabot says, "as it descends and allows the lung to take its place." This "peeling off" of the thick diaphragm and its replacement by the line of lighter lung tissue can be seen, in a proper position and light, as a shadow moving down the lower part of the chest wall.

In order to perceive this phenomenon, the patient must lie flat on his back, his entire chest bared, his feet turned toward the only source of light in the room, the observer seated at his side, and must take a full breath. The shadow, of course, depends upon the ripple of different level that passes over the chest surface as the thick diaphragm recedes and gives place to the thin lung tissue.

In pleuritic adhesions, the movements of the diaphragm are prevented; in effusions, emphysema and pneumonia, the diaphragm is sepa-

rated from this chest wall, hence the shadow is not seen. Solids or fluids under the diaphragm, unless extremely large, do not interfere with its movements.

TYPES OF BREATHING.

The natural type of breathing is that which starts with the diaphragm, the first perceptible movement being an obtrusion of the abdomen. Thence the process spreads upward, involving the ribs and shoulders. This type is called the **abdomina-costal** type, and is almost universal among men. In civilized women, on the contrary, the diaphragm takes but little part in respiration and the ribs in consequence acquire an exaggerated importance. This is called **costal breathing.** Investigation demonstrates that it is due to a difference of dress, and not to genuine sexual differences. The types may be, and often are, modified by disease.

Abdominal Breathing: In disease of the chest, where costal expansion causes pain, as in pleurisy, pneumonia, etc., and where intra-pulmonary pressure is increased, as in hydro- or pneumo-thorax and emphysema, the diaphragm is the main factor in respiration, producing the abdominal type of breathing.

Costal Breathing: In abdominal lesions, on the contrary, such as tumors, ascites or visceral

enlargement impeding the movements of the diaphragm, the breathing is mostly costal.

In paralysis of the phrenic nerve, the breathing is, of course, costal, and in paralysis of the intercostals, the reverse. The latter is, however, rare, except immediately preceding death.

Rapid Shallow Breathing: It is probable that the vagus nerve which supplies the lungs contains both inhibitory and accelerator fibres, regulating the rhythm of the heart. Collapse calls the accelerators into play, distension the inhibitors.

The breathing, therefore, is rapid and shallow in any condition which limits the expansion of the lungs, as in pneumonia, from consolidation of lung tissue, atelectasis, from alveolar collapse, pleurisy, owing to the pain induced by inspiration, and emphysema, due to the exaggerated intrapulmonary pressure.

Doubtless the increased frequency of breathing in fevers, especially infectious and low-grade fevers, where the metabolism is rapid, is due in some measure to the accumulation of CO_2 in the blood, acting as an abnormally powerful stimulus to the respiratory center. It would seem, however, that this center, like most others, can be over-stimulated, for CO_2 narcosis in such fevers slows and enfeebles the respirations and finally paralyzes them.

The same remarks apply to cases of respiring air poor in oxygen and rich in CO_2.

Slow, Shallow Breathing: It is to be remembered that, in accordance with Nature's compensatory provision, no matter how serious the condition, as long as the vital processes of the body remain intact, shallowness of respiration is always compensated for by increased frequency. When the breathing is both shallow and infrequent, it indicates that this compensatory power of the system is failing—a very grave condition. It ocurs either in profound narcosis (morphine poisoning) or as a precursor of death, when dissociation of the vital centres has already begun.

PRESSURE CONSIDERATIONS.

Since the lungs are in communication with the external atmosphere, it follows that the air pressure in the lungs at any time of rest, whether at the end of inspiration or expiration, is equal to that of the atmosphere. When the lungs are expanded in inspiration, their capacity increases more rapidly than the air coming through the narrow glottis can keep pace with, and there is a temporary fall in pressure until the pause at the end of inspiration. Contrariwise, the recoil of the chest is a little too rapid for the outgoing air to equalize,

and there is a rise in pressure. This variation, however, is in normal respiration very slight.

In the part of the thorax outside the lungs, on the other hand, since it is protected from atmospheric pressure from outside by the chest wall, and that which reaches it through the lung-sac is modified by the elastic recoil of the lung, the pressure is always negative. The more the thorax is enlarged, stretching the lungs with it, the more forcibly this elastic recoil brought into play and the more negative becomes the intra-thoracic pressure. It may at any given moment be represented as that of one atmosphere less the elasticity of the lungs.

Emphysema: Any influence which interferes with the free expulsion of air through the trachea increases the intrapulmonary pressure, and opposes extra resistance to the elastic recoil of the lungs, bringing into play the muscles of forced expiration to drive out the air. If this intrapulmonary pressure is kept up for any considerable length of time, the elasticity of the lungs at last becomes seriously impaired and they are no longer properly emptied. As a consequence they are in a constant state of more or less inflation which is known as emphysema. The same result occurs from any pathological process in the alveoli them-

selves, which impairs their elasticity; also physiologically in old age.

This condition is seen notably in chronic bronchitis and bronchial asthma.

Vicarious Emphysema is seen in parts of the lungs where the other portions are temporarily or permanently incapacitated, or in one lung where the other is out of commission. The extra duty entailed upon the sound area causes an undue inflation and retention.

Cardiac Involvements: Sooner or later, of course, the extraordinary and constant expansion of the lung causes a backward pressure upon the pulmonary artery and interrupts the cardiac cycle, mechanically and also by virtue of inadequate oxidation of the blood. This is compensated for a time by hypertrophy of the right ventricle, but eventually compensation is broken and stasis ensues.

Pneumothorax: In tuberculosis and other degenerative processes, the pleural sac occasionally ruptures under slight exertion and air is forced into the thoracic cavity at each inspiration. Or the same result is produced by a penetrating wound of the chest, admitting air from outside. This, of course, immediately raises the intrathoracic pressure to that of the atmosphere, and as this entrapped air does not escape as readily as it enters, the pressure is

soon raised above that of the lungs, embarrassing their expansion and the heart action, and causing death if not relieved.

Coughing: Any attempt at expiration with closure of the glottis, of course, raises the intrapulmonic pressure. A conspicuous and extreme example is seen in the familiar phenomenon of coughing. The irritation of the tubes causes a spasmodic closure of the glottis; at the same time a violent effort is made at forced expiration, raising the pressure considerably and resulting in the explosive opening of the glottis and expulsion of air under high pressure, which constitutes the noisy "cough." During the period of compression, pulmonary circulation is interfered with, general venous stasis occurs, and if long continued causes cyanosis, and occasionally rupture of one of the vessels.

Coughing results from any condition which (1) irritates the upper respiratory tract, as in laryngitis, tonsillitis, pharyngitis, bronchitis, etc., (2) increases the intrapulmonic pressure, thus inducing the patient to attempt a forcible expulsion of the excessive air, as in emphysema, asthma and long-continued bronchitis, where air has accumulated in the lungs, and (3) interferes seriously with the exchange of gases between the blood and the atmosphere, thus pro-

ducing a sort of air-hunger in the lungs, of which the most familiar example is seen in pneumonia.

It should be borne in mind that these three causes of cough may co-exist in the same patient.

Venous Stasis (Cyanosis): The large veins above and below the thorax are subject to atmospheric pressure; the venae cavae and right auricle, on the contrary, are under pressure of less than one atmosphere. This difference in pressure levels acts as a suction influence, assisting the flow of venous blood to the heart. It is, of course, more marked with every expansion of the lung in inspiration, which makes the pressure still more negative. In pneumo- and hydro-thorax, of course, this suction phenomenon is lacking, because the intrathoracic pressure is raised to the level of, and sometimes even higher than, the intrapulmonic. This helps to explain the venous stasis occurring in these conditions.

Insufficient Oxidation: Experiment shows that the relative pressures of oxygen in the alveolar air, where the exchange is made, and the venous blood that is brought to the lungs by the pulmonary arteries, are as follows:

Alveolar air—23 to 129 mm.

Venous blood—40 mm.

It will be readily seen that any diminution in the oxygen pressure at the lungs will result in an abnormally small impartation of this gas to the venous blood, and consequently in poor oxygenation of the tissues. This may occur either from (a) poverty of the surrounding air in oxygen, or (b) any condition of the respiratory mechanism which tends to rarify the air as it enters the lungs—e. g., stenosis of the trachea or bronchi—causing undue negative pressure during inspiration; or (c) emphysema, in which the inspired air is continually diluted by an excess of residual air.

CO_2 Narcosis: Any interference with the process of respiration, unless compensated for, will ultimately result in an accumulation of CO_2 in the tissues to an extent which will render them inert and unable to perform their proper functions. This condition is known as CO_2 narcosis, and is the cause of death in all diseases which kill by impeding respiration.

Variations in Respiratory Frequency: From what has gone before, it is evident that the rapidity of respiration may be modified by either one of two general conditions:

(1) By any condition influencing the expansion and recoil of the lungs, acting as a stimulant of the accelerator or depressor nerves of the lungs.

(2) By any condition influencing the quantity of carbon dioxide in the blood, acting as a direct stimulant of the respiratory centre.

It is a well-known fact that the respiration is relatively rapid in infancy, 35 to 40 per minute, gradually diminishing in frequency until in adults it remains pretty constant at 18 to 24 per minute. This is doubtless due in large measure to the relatively smaller available lung capacity in children, which increases in greater ratio than the growth of the body; and to the relatively greater metabolic activity of the tissues in childhood consequent upon growth.

Increased Frequency: Undoubtedly the increased frequency of respiration in fevers, as already explained, is largely due to accumulation of carbon dioxide.

Pleurisy causes rapid respiration because the limited expansion of the chest due to the pain sets in motion the reflex mechanism of the vagus accelerators.

Pneumonia, phthisis, etc., exhibit rapid breathing because of the small amount of available lung area operating both by way of the vagus and by the accumulation of CO_2 in the blood.

In hydro- and pneumo-thorax the movements of the lungs are limited by the pressure in the thorax; here both the vagus reflex and

the excess CO_2 in the blood operate to increase the frequency of respiration.

In emphysema and atelactasis the excess CO_2 appears to be the causative factor.

In paralysis of the phrenic nerves (innervating diaphragmatic respiration) or paralysis of the costal nerves (innervating thoracic respiration) the expansion of the thorax is much limited, hence the pulmonary branch of the vagus is stimulated and the group of motor nerves which are intact call their mechanism into increased play to compensate for the inactivity of the other group.

Many of the causes of increased respiratory frequency only manifest themselves when some extra demand is made upon the respiration, such as exertion, which in health would not embarrass the breathing at all. Especially is this the case in those conditions where it depends upon an insufficient oxidation and compensated heart lesion.

In chronic lesions which would otherwise tend to induce rapid breathing, the process may be so gradual that the system adapts itself to the disability. In tuberculosis, for example, the lessened lung area is frequently offset by the reduction of body weight, thus lessening the necessity for oxygen and CO_2 elimination.

Diminished Frequency: This is practically always due to brain compressions or toxic paralysis of nerve centres by toxines or drugs, e. g., uremia, morphine poisoning, etc.

In all those cases where increased frequency is caused by accumulation of CO_2 in the blood, this condition if prolonged sufficiently to induce CO_2 narcosis, will eventually slow the respiration.

Variations in Rhythm are quite common in health. It is difficult to assign any definite or simple explanation for the physiologic irregularities that are frequently seen, sometimes even in sleep, especially among children and nervous people. It is highly probable that they depend largely upon central and reflex disturbances of the inhibitory and accelerator factors of the nervous mechanism, of the same kind, only differing in degree, as those inhibitions of the centre which arise from powerful emotions and cause prolonged variations in rapidity.

Cheyne-Stokes Breathing is the most notable example of pathological abnormality in respiratory rhythm. In this type of breathing there is a rapid crescendo and diminuendo, both as to frequency and depth, the paroxysms lasting from thirty seconds to a minute, and intervened by a pause in respiration. The

cause of the phenomenon is unknown, but it doubtless has its explanation in some disturbance of the central nervous system. It is usually of grave import. In children, however, it occasionally occurs physiologically during sleep.

Cabot describes a very irregular gasping respiration, accompanied by a nodding of the head, "the chin being thrown quickly upward at each inspiration, and falling slowly during expiration," which he attributes to nervous dissociation, and regards as a precursor of death. This is seen in uremia, and certain diseases of the brain.

Asthma is a common illustration of disturbed rhythm; the exciting cause is usually reflex, as described under Reflex Inspiration.

Variations in Amplitude or Depth are quite common within physiological limits, depending upon practically the same conditions that determine the rapidity of the process.

Dyspnea: In a general way, of course, anything which in any way whatever interferes with any of the processes involved in respiration produces dyspnea; conversely, dyspnea may be a sign of any one of these interferences. In practice, however, the term is used only in connection with actual labored breathing, i. e., breathing against extraordinary pressure, and

in this restricted meaning dyspnea is practically always due either to (1) air pressure in the lungs or the thorax, or (2) venous stasis, caused by a backing up of the circulation in the lungs, as in pneumonia, valvular diseases of the heart, kidney disease, etc. There is another form of dyspnea due to air-hunger, in anemia, advanced tuberculosis, and wasting diseases, but it is rather a gasping phenomenon than true dyspnea.

The cardiac causes of dyspnea will be found explained in the section on The Vascular System.

THE VASCULAR SYSTEM.

Anemia: The essential morbid element of anemia is a diminution of the number of red corpuscles in the blood, associated with important abnormalities in the character of these corpuscles. Sometimes the number is scarcely lessened at all, but the amount of hemoglobin contained by them is greatly reduced. Nucleated red cells are usually formed in great quantities. Some of these are undeveloped forms of red blood corpucles, doubtless due to Nature's attempt to repair the rapid destruction of red corpuscles by hurrying out new ones. Others (megaloblasts) resemble blood plates, but are considerably larger. Their significance is unknown.

Anemia, therefore, points to one of two general classes of causes, (1) a disease of the blood itself, interfering with the proper manufacture and development of red corpuscles, or (2) a condition which attacks the red corpuscles so as to rob them of their hemoglobin. Anemia of the first type is known as primary, and of the second variety as secondary anemia.

In primary anemia, there are, of course, many defective, diseased blood-cells found in the field, and both cell-count and hemoglobin percentage are exceedingly low. In secondary anemia, the cells are diminished in number and poor in hemoglobin, but not nearly to the extent of primary anemia, and there are immature cells in the field, but not morbid or defective forms.

The underlying causes of primary anemia (pernicious anemia) are not well understood. Secondary anemia, which is far commoner, indicates usually some chronic infection, in which the toxins are hemolysing the cells and destroying them, as in cancer, nephritis, tuberculosis, malaria, gall-bladder infection, suppurative diseases, and focal infections of all kinds.

Certain drugs have marked hemolytic action on the blood, notably ammonia and chloroform. So also have numerous toxins, of which the venom of the snake is a conspicuous example.

To this toxic hemolysis must be attributed most of the secondary anemias resulting from infectious diseases and malignant growths.

BLOOD PRESSURE.

Velocity and Pressure: Two elements enter into the mechanical features of circulation, namely, the velocity of the blood flow, and blood pressure. The former represents the amount of force that is expending itself in the direction of the axis of the vessel, driving the blood along it and may be measured at any point by inserting a bent graded tube parallel to the axis of the vessel and noting the height to which the blood arises. The latter represents the force expended in a direction at right angles to the axis of the vessel, and may be measured at any point by inserting a tube at right angles to the axis and noting the height to which the blood rises.

Factors: The factors which take part in and modify these mechanical phenomena are (1) the heart beat, (2) the friction in the vessels, (3) the resistance of the vessel walls, (4) the constriction or dilatation of the vessels by vasomotor influences. Of late years it has been discovered that certain of the ductless gland secretions (endocrines) play an important part in the blood pressure. The secretions of the adrenal glands, and of the gonads (ova-

ries and testicles), tend to raise the pressure, while that of the thyroid gland tends to depress it. Just how these secretions bring about their effect upon the blood tension is not definitely known; probably through the vaso-motors.

High Blood Pressure results from any pathological condition which (a) increases the volume of blood in the vessels; (b) increases the force of the heart beat; or (c) offers abnormal resistance to its passage. Conditions which increase the total volume of blood are rare, the increase usually occurring either on the venous or arterial side, at the expense of the other.

Simple Cardiac Hypertrophy throws an abnormally large quantity of blood into the aorta at each systole, at the expense of the veins, thus raising arterial and lowering venous pressure. The impetus with which the blood is propelled also contributes to the increase of pressure.

Drugs and toxins of certain kinds exaggerate the force of the heart and thus raise pressure. Most of these agencies also cause constriction of the vessels, which contributes to the increase of pressure.

Nephritis raised blood pressure primarily by vaso-constriction in the kidney, thus increasing peripheral resistance, and secondarily by toxic

sclerosis of the artery walls, abolishing their expansibility.

Arteriosclerosis increases the rigidity, hence the resistance of the vessel walls, and thus raises pressure.

Excessive activity of the adrenal or gonad glands, or deficient secretions of the thyroid, or both, will produce an elevation of blood pressure.

Low Pressure: Conversely, any influence which (a) weakens the heart beat, (b) lessens the volume of the blood, or (c) diminishes resistance to its passage, lowers pressure.

Myocarditis, Dilatation and **Fatty Degeneration** of the heart are attended with low arterial pressure, both because of diminished impetus and because an abnormally small quantity of blood is thrown into the aorta at each systole.

Hemorrhage lowers pressure by taking blood out of circulation.

Low Fevers (typhoid, typhus, etc.) and **Constitutional Diseases** which inhibit nutrition without any sclerotic changes in the blood vessels, rob the vessel walls of their tone, thus abolishing their elastic recoil and lowering pressure.

Excessive or **Perverted Activity** of the **Thyroid Gland,** or excessive deficiency of the secretions of the adrenal or gonad glands, or both, will bring about a low blood pressure, and is a frequent cause of this condition, especially in females.

PULSE.

The Pulse: If the vascular system consisted of a set of rigid tubes, each discharge of blood from the ventricle would push the whole mass of blood forward and simultaneously empty from the venae cavae into the auricle a similar quantity of venous blood. The elasticity of the aorta wall makes it easier to distend the artery than to move the whole mass of blood forward, and this is what happens primarily to make room for the blood discharged by the heart beat.

As soon as the semi-lunar valves close, the elastic coat of the aorta recoils, and drives the columns of blood onward. This succession of distension and recoil passes down the entire arterial system, in the form of a wave, diminishing as the total area of the channel increases, until in the capillaries, where the total area suddenly increases many times, the wave is lost. This wave of distension and recoil constitutes the pulse, and for the reason described is only seen in the arteries.

Abnormal Pulse: The normal propagation of the pulse wave may be interfered with by (a) conditions within the heart, or (b) conditions in the vessels. Such interference may make itself manifest in (a) the amplitude, (b) the regularity, (c) the velocity of the wave. These factors constitute the character of the pulse. The pressure in the vessel is frequently included in the character of the pulse, but is really an entirely different phenomenon from that of the pulse proper, pressure being a static condition at any given moment, while the pulse is a dynamic condition requiring time as one of its elements.

Increased Amplitude: In general, anything which increases the force of the heart beat increases both the amplitude and velocity of the pulse wave. Hypertrophy of the heart, especially of the ventricle, in its early effects, is one of those influences. Later, when compensation fails, the amplitude of the wave is diminished and generally the velocity also. But it must not be forgotten that the effect of increased heart force on the wave may be offset by conditions of the vessels.

Diminished Amplitude: Myocarditis and fatty degeneration weaken the muscular force of the heart, and therefore diminish both amplitude and velocity of the pulse wave, unless offset by vascular conditions.

In **aortic stenosis,** the amount of blood thrown into the artery is smaller than normal, in spite of the hypertrophy of the ventricle, hence the amplitude of the pulse wave is reduced. The smallness of the pulse in contrast with the force of the ventricle, is very characteristic of this lesion.

In **mitral stenosis,** the amplitude and velocity of the pulse wave is diminished because of the less quantity of blood poured into the ventricle at each systole.

Mitral Insufficiency, as long as compensation is good, has no perceptible effect upon the pulse wave, as the disturbances are all back of the ventricle.

Aortic Insufficiency has a very characteristic effect upon the pulse wave. The hypertrophied condition of the ventricle augments the force behind the pulse and increases its amplitude, giving a high incline of ascent, but the decreased resistance offered by the leaky valve to the aortic recoil induces a rapid, almost sudden collapse of the artery, giving a steep incline of descent. This excessive backward collapse may even be seen in the capillaries, evidenced by capillary pallor at each systole. This pulse is called the **Corrigan** pulse, and is pathognomonic of aortic insufficiency. **(Quincke Pulse.)** It may, of course, be absent in this

lesion, if conditions of the vessels offset the valvular influence.

Failure of Compensation: In all of the valvular lesions, as soon as compensation fails, all of the characteristic effects of the compensated lesions disappear and the effect common to them all becomes one of enfeebled heart force, namely- small amplitude and low velocity.

Catacrotic Pulse Waves: The elasticity (elastic recoil) of the arterial walls produces some modifications of the arterial pulse wave just described, by means of reflected waves set in motion by the recoiling wall of the artery. These are called **catacrotic waves.** The most significant of them is the **docrotic wave,** which occurs immediately after the recoil has started, and noticeably interrupts the downward incline of the pulse. It is caused as follows: When the aorta begins to recoil it propagates the blood in both directions, toward the peripheries and toward the heart. The latter organ has shut its semi-lunar valves against which the proximalward wave strikes and is reflected as a distalward wave, once more producing a slight arterial distension.

Exaggeration and Diminution: In extremely rigid conditions of the artery walls, as in arterio-sclerosis and atheroma, the effect of the secondary wave is, of course, hardly percep-

tible, and in these conditions, therefore, there is a notable absence of the dicrotic wave. The same is true when the blood in the artery is under high pressure, so that the artery wall is already, by virtue of the fundamental wave, under tension, hence in cases of high arterial tension the dicrotic pulse is diminished. On the other hand, an easily distensible artery and a low blood pressure favors the perceptible effect of the secondary wave, and in these conditions of dicrotic wave is very noticeable—e. g., typhoid fever, tuberculosis, malignancy, etc.

In **Aortic Insufficiency,** of course, the dicrotic pulse is absent, because of the recoil of the artery the blood, instead of striking the semilunar valves and producing a secondary wave, passes through the leaky valves into the ventricle. The collapse of the pulse, as already stated, is sudden, with no catacrotic wave.

Heart Block: In rare instances a pathological condition is seen consisting in an interruption, by means of a tumor, gumma, degeneration, or other destructive process, in the muscular isthmus between the aruciel and ventricle (the Bundle of His). The functional aberration resulting from this lesion is a disconnection of the auricle and ventricle in regard to their rhythmicity, each maintaining

a rhythmic beat of its own, and is known as heart block. Sometimes the block is complete, in which case the rhythmicity of the auricle and ventricle bear absolutely no relation to each other; in others the block is only partial, and the respective rhythmicities then bear some regular proportion to each other, e. g., the ventricles will beat twice to the auricle's once.

HEART ACTION.

The contraction of the heart muscle, of course, diminishes, and probably in health completely obliterates the cavity of the ventricle, driving the contained blood into the artery. The change in the form of the ventricle thus produced differs with the position of the heart, i. e., with the position of the body, but in general it is decreased in its vertical and transverse, and increased in its dorso-ventral diameters. The spiral arrangement of the superficial muscles causes a rotation of the ventricles in systole. This rotation compensates for such vertical shortening as occurs, and maintains the apex of the heart in its normal position against the chest wall, while the sudden contracting of the heart muscle produces the apex impulse. The impulse is also augmented by the sudden straightening of the curved aorta by its distension with blood.

Exaggerated Apex Beat: When from any cause, such as increased resistance or forced acceleration, the heart muscle is hypertrophied, one of the earliest and most characteristic manifestations of its hypertrophy is an exaggeration of the apex beat against the chest wall, especially in cases (most common) where the hypertrophy either begins in or is confined to the ventricles.

This phenomenon is seen in those valvular diseases which produce ventricular hypertrophy, viz., insufficiency and stenosis of the semi-lunar valves, as well as in so-called idiopathic hypertrophy.

Heart Sounds: The heart beat is accompanied by two sounds, corresponding to the ventricular systole and diastole, succeeded by a pause corresponding to the period of rest. The first sound is a dull booming character, the second of a sharper tone, and a minor third higher in pitch. The following occurrences are synchronous with the first sound and probably enter into its composition: (1) Closure of the auriculo-ventricular valves. (2) Contraction of the heart muscle. That the first of these elements is not the sole factor is proven by the occurrence of the first sound in a bloodless beating heart, and by the booming nature of the sound. The second sound is doubtless caused by the closure of the semilunar valves,

as it disappears on hooking back those valves in the living heart.

Careful experiment has shown that the first sound occurs at the beginning of the systole, and the second at the end of the systole (diastolic).

Exaggeration of the First Sound: In hypertrophy of the heart muscle in the early stage, the hypertrophy is accompanied by an exaggeration of its contractibility, manifested by an exaggeration of the muscular element of the first sound. Later, when compensation begins to fail, this gives place to an enfeeblement of the first sound, which, however, retains a more booming character than normal because of the greater volume of muscle involved.

Perverted Heart Sounds in Valvular Disease: Diseases of the heart involving the valves, both leakage and stenosis, give rise to characteristic abnormalities in the heart sounds which call for special explanation and interpretation.

Mitral Insufficiency: The mitral valves close at the beginning of the ventricular contraction, to prevent any of the blood contained in the ventricle being driven back into the ventricle. If the mitral valves are "insufficient," i. e., if they leak, at each ventricular systole some of the blood is driven back into the auri-

cle (**regurgitation**). This backward current, meeting the oncoming stream in the auricle, causes a whirl of blood, which, impinging on the edges of the valves, produces a peculiar blowing sound, called a **murmur.** This murmur, of course, occurs at systole, and is a systolic murmur.

If, on the other hand, the mitral orifice, from any cause, such as thrombosis or inflammatory vegetation, is stenosed, the passage of the blood from auricle to ventricle is impeded, and the rush of liquid under increased tension through the narrow orifice produces a rippling sound, never so loud or blowing as in a mitral leakage.

This sound, of course, occurs just prior to ventricular systole, and is called a **presystolic murmur.**

Aortic Insufficiency: When there is a leakage of the semilunar valves of the aorta, at each recoil of the aorta the blood is partially driven back into the ventricle and, meeting the oncoming stream from the auricle, produces a sound similar to that produced in mitral leakage, but it is, of course, heard following systole, and is called a **diastolic murmur.** The first heart sound is indistinct (though loud) because of the overdistension and slow contraction of the ventricle.

Aortic Stenosis: In stenosis of the aortic orifice the blood is forced from the ventricle under increased pressure through a very narrow opening, and a sound is produced similar to that described in mitral stenosis. It occurs, of course, during ventricular contraction, and is a systolic murmur.

The sounds produced by the lesions above mentioned are propagated along the direction of the blood current and as the valves themselves all lie within a very small area the sounds are best differentiated by listening to them at the suburban heart points, to which they are transmitted. Thus the sounds and their significance may be classed as follows:

(1) **Systolic Murmur,** heard best at apex, indicates mitral insufficiency.

(2) **Pre-systolic Murmur,** heard best at apex, indicates mitral stenosis.

(3) **Diastolic Murmur,** heard best at aortic arch, indicates aortic insufficiency.

(4) **Pre-diastolic Murmur,** heard best at aortic arch, indicates aortic stenosis.

Pulmonary Lesions: The valves of the right heart are subject to precisely the same conditions as here described, and present a corresponding set of sounds, but their occurrence is so rare as to demand no especial attention.

Haemic Murmurs: Similar dynamic conditions to those of valvular insufficiency may be brought about by abnormalities of the blood, which decrease its density, and murmurs are frequently thus produced. Particularly is this the case in anemia. The sounds are neither so loud nor so constant as in valvular lesions, and are called haemic, or functional murmurs.

Accentuation: Anything which augments the force of the heart beat increases the muscular impetus and therefore accentuates the first sound of the heart. We have already seen that hypertrophy is the chief of these influences; exercise, emotions, and certain drugs also have this effect, and it is not an infrequent practice among diagnosticians to administer strychnin in order to augment the first sound and bring out suspected abnormalities.

The second sound, depending upon the closure of the semilunar valves, is accentuated by anything which increases the intra-arterial pressure, and its accentuation is pathognomonic of disease in which peripheral resistance is increased, of which arterio-sclerosis and kidney diseases are familiar illustrations. Accentuation of second sound is, of course, physiologic in old age, because of atheroma of the vessels.

Accentuation may occur from the causes described in any of the valves separately, which

together make up the respective sounds. These separate accentuations must be diagnosed by auscultating at the outlying points of sound transmission for the separate valves.

Reduplication of the heart sound is due to the asynchronous occurrence of the events which produce it. In the first sound, of course, the valvular element is the only one which can be duplicated and as this element is completely overshadowed by the muscular element, its redupilcation is practically unrecognizable.

Reduplication of the second sound is not an infrequent symptom and is usually due to some pathological condition of the coronary arteries. The two sides of the heart, being equally nourished, do not functionate synchronously; the semilunar valves close asynchronously, and give a double sound.

The same phenomenon results from an unequal tension in the two ventricular cavities, due to valvular leaks and stenoses.

Pseudo-Accentuation: Myocarditis and fatty degeneration reduce the force of the muscle contraction and therefore make the first sound weak and indistinct. The second sound, by contrast, seems accentuated; but in cases of high blood pressure from other causes, the second sound may, of course, be genuinely reduplicated.

Perverted Heart Cycle in Valvular Disease: Just as valvular diseases of the heart give rise to certain characteristic perversion of the heart sounds, so do they bring about certain characteristic interferences with the heart cycle, evidenced by dyspnea, cyanosis, pulmonary congestion, etc. Their explanation is as follows:

Mitral Insufficiency: Thus, if the mitral valve leaks, at each ventricular contraction a portion of its contained blood in forced back into the auricle and an insufficient quantity pumped into the aorta. The auricle is then receiving blood both from the venae cavae and from the ventricle and becomes much distended, and the tendency is for a backward stasis of circulation. However, the auricular muscle responds to this demand by hypertrophying (compensatory hypertrophy) and contracting more forcibly. In time, of course, hypertrophy can no longer make up for the increased work, and compensation fails. The result, as one would suppose, is backward stasis in the veins, high venous and low arterial pressure, the former causing dropsy and CO_2 poisoning, the latter insufficient oxygenation, shortness of breath and general atony. (See Respiration and Elimination.)

Aortic Insufficiency: Leakage of the semilunar valve produces, by the same dynamic

process, a compensatory hypertrophy of the ventricle and eventually backward stasis.

Mitral Stenosis: Stenosis of the mitral orifice, although a reverse condition to insufficiency produces the same results by a somewhat different mechanism. Here the narrowness of the opening imposes a systolic pressure upon the auricle, which is compensated by auricular hypertrophy, later producing backward stasis.

Aortic Stenosis: Narrowing of the aortic opening operates upon the ventricle precisely as vertical stenosis does upon the auricle, with the same backward train of events.

Leakage and Stenosis of the Right Heart: Insufficiency and stenosis of the valvular mechanism of the right heart produces a set of conditions precisely corresponding to those described for the left heart. In these cases, however, the pulmonary circulation is the first to feel the effects of backward stasis due to failing compensation, and respiratory difficulties are the earliest and most direct results. Fortunately, as already stated, they are much rarer, owing to the less opportunity for functional derangement than in the systemic circulation.

The Coronary Arteries: Another source of interference with the cardiac cycle is frequently

seen in a lesion of the coronary arteries. In order to properly carry out their function all the heart muscles must themselves be regularly and adequately supplied with nutrient blood, and any condition of the coronary arteries—e. g., sclerosis, embolism, thrombosis—will produce a disturbance in the performance of the heart cycle. Sudden stoppage of the heart (in diastole, of course) often results from this cause, which is also thought to be the explanation of the phenomenon known as **angina pectoris.**

Intrapulmonary Pressure: Any condition producing an increase of intrapulmonary pressure will, if continued long enough, embarrass the right ventricle, and bring about its hypertrophy, with eventual failure of compensation and fatal stasis. Emphysema is a notable example of this. (See Respiration.)

Systemic Pressure, long continued, such as is caused by chronic Bright's disease, diabetes, arterio-sclerosis, etc., will bring about the same train of results in the left heart.

DIGESTION.

Dysarthria (Difficulty in Chewing): One of the earliest manifestations of bulbar paralysis is dysarthria, or difficulty of jaw movement, due to involvement of the root of the fifth

nerve. The lesion of this particular part is probably no earlier in fact than that of other bulbar areas, but its impairment is noticed first. Later, there is complete inability to masticate. This condition may also be due to arthritis affecting the angle of the jaws.

Pytalism is an excessive secretion of saliva. It is rarely, if ever, a primary complaint, but depends upon some other pathological condition, and the saliva is usually altered in character as well as increased quantity.

Inflammations of the **Mouth** and **Throat,** unless accompanied by a high temperature, are always attended by an increased flow of saliva, due partly to vaso-dilator conditions and partly to increased reflex stimulation. In such cases it is usually acid in reaction, because of the increased absorption of CO_2 and other metabolic acid products.

Undoubtedly the pytalisms of inflammations of the mouth and tongue are partly due to irritation of the afferent fibers of the glosso-labio-laryngeal nerves.

Gastric Pytalism, as seen in catarrhal gastritis, gastric ulcer, before vomiting, etc., are due to stimulation of the afferent fibers in the gastric branch of the vagus.

Pregnancy is attended by a more or less degree of pytalism.

Mercurial Pytalism is due to hyperstimulation of all the salivary glands by the drug.

In **fevers** the watery part of the saliva is rapidly absorbed by the mucous membrane to compensate for the general anhydrous condition of the tissues and the secretion is therefore thick and viscid, and feels dry and sticky. The same condition is found, and for the same reason, in **diabetes** and certain forms of **nephritis.**

Acid Saliva: The reaction becomes acid in fevers, diabetes, gout, rheumatism and nephritis, because of the absorption of metabolic acid products, chiefly CO_2 in the first two diseases and uric acid in the others. In gouty subjects the acidity is sometimes so high as to erode the chin and corners of the mouth.

Inasmuch as the action of pytalism is favored by an alkaline medium and inhibited by acids, it is easy to understand the poor diastatic quality of the saliva in fevers, diabetes, rheumatism, gout, nephritis, in which the reaction of the secretion is acid from containing CO_2 and uric acid.

Dry Mouth is a condition, described first by Hutchinson, in which the secretion of the saliva is inhibited as the result of a central nervous disturbance. The parotid glands become hard but painless.

In fevers the watery portion of the saliva is rapidly absorbed by the mucous tissues to compensate for the general anhydrous condition of the body, and the saliva is therefore thick and viscid.

Dysphagia (difficulty in swallowing), of course, results from any impairment of any of the above described factors in deglutition, or from any condition making the performance of their part in the act painful.

Inflammation of any portion of the alimentary tract from the mouth to the stomach, including glossitis, pharyngitis, tonsillitis and esophagitis, make swallowing difficult because of the pain caused by the contact of the food and by muscular contractions.

Ulcers, Growths, etc., have the same effect for the same reason.

Spasm of the Passages offers an obstacle to swallowing, both by closing the passage and by temporarily paralyzing the musculature. In spasms, occurring during the act of deglutition the food is immediately regurgitated.

Bulbar Paralysis results in voluntary dysphagia due to involvement of the medullary root of the twelfth cranial nerve, one of the most distressing symptoms of this lesion. The same lesion, of course, paralyzes the reflex mechanism of the second and third stages of

deglutition, but these are subordinate to the first or voluntary stage.

Choking: In bulbar paralyses and central lesions involving sensory paralysis of the laryngeal nerves, the stimulus which protects the laryngeal opening at the glottis is wanting, and particles of food frequently enter the larynx and cause partial or complete asphyxia.

Fermentation and Flatulence: When the motor mechanism of the stomach is so interfered with as to delay the passage of food through it, the carbohydrates of the food are fermented by the action of the bacteria of fermentation, forming organic acids (lactic, butyric, acetic). These in turn form acid gases, that are eructated through the mouth. The occurrence of these fermentations is shown by the establishment of the hyperacidity of the stomach due to excess of the acids mentioned.

The conditions which most markedly diminish the motility of the stomach and thus produce fermentation are dilation, pyloric obstructions, malignant growths, and chronic gastritis.

Excessive food may also interfere with the muscular movements of the stomach, and produce the above train of symptoms, except that in such case the acidity of the stomach contents will be relatively below par, as enough is not secreted to take care of the excessive amount of food.

Digestibility of food is usually gauged, so far as the stomach is concerned, by the length of time it remains in the stomach, and as it is discharged into the duodenum as soon as it is liquefied, this is equivalent to estimating it by the ease with which the stomach reduces the food in question to a fluid.

Atony of Stomach Walls: Due to distension, (1) putting greater strain on muscles, (2) compromising the vessels and lowering nutrition, and (3) dragging organ downward and kinking the pylorus.

Pyloric Stenosis, producing first hypertrophy and late dilatation of muscular walls.

Defect in Nutrition of stomach walls.

Disease of Stomach Walls—gastritis, carcinoma, etc.

Passive Congestion of Stomach, interfering with nutrition.

"Splashing" Sounds: As already stated, the normal stomach is always distended in exact proportion to the amount of solid or liquid food it contains, its muscular coat contracting tightly around these contents and promptly expelling all air and gas that may be present or formed. If the muscular walls be atonic, however, the stomach will sag under the weight of liquid, gas and air will be present,

and upon shaking the patient the contents may be heard to "splash." This is pathognomonic of gastric atony. Its exhibition in patients apparently free from stomach troubel is explained by the fact that in early stages of atony the stomach is still able to empty itself regularly, and therefore gives no signs of gastric indigestion.

Pyloric Insufficiency is seen sometimes in muscular diseases and malignant infiltrations, the contractile power of the pylorus being lost and the food pouring through the orifice too quickly. Its results are, of course, intestinal indigestion; one of the most noticeable symptoms being a regurgitation of gas produced by the action of the alkaline juices of the duodenum on the acid chyme.

Eructation is a motor reflex, mediated as to its afferent stimulus either by the sensory fibres of the gastric vagus in the stomach itself, or by the glossopharyngeal nerves.

Vomiting is a reflex, having its centre in the medulla (q. v.). Its afferent paths are numerous. Brunton enumerates them as follows: Pharyngeal branches of glosso-pharyngeal; pulmonary and gastric branches of vagus; gastric branches of splanchnic; renal, mesenteric, uterine, ovarian and vesical nerves. Its efferent paths are the motor fibres of the gastric vagus and splanchnic.

The centre is amenable to influences direct from the brain (central vomiting); and to those of certain conditions of the blood, as in infectious diseases.

Paresis of the Stomach Walls is a very rare occurrence, but is occasionally met with. The result is an acute dilatation of the stomach, due to the complete abolition of the "tonal" energy.

Spasm of the Cardia and Pylorus is a reflex phenomenon, due to irritation of the stomach nerves by hyperacid contents. It produces what is known as pneumatosis, the gas in the stomach being caught between the two spasmodic contractions and distending the stomach, causing great pain.

Dyspnea and Cardiac Failure: Any sudden or serious distension of the stomach may produce dyspnea by undue pressure on the diaphragm and faintness and palpitation by interference with the dynamics and innervation of the heart.

Any condition which irritates the gastric nerves can disturb the heart and respiration reflexly through the vagus nerve. This is especially common in hyperacidity of the stomach.

Anacidity: In acute and chronic gastritis in which there is excessive mucous secretion, the activity of the cover cells is interfered with

and a deficiency in hydrochloric acid results. The cause here is local. Later there is atrophic or cirrhotic destruction of the gland cells, resulting in complete failure of HCl and pepsin.

In **Anemia, Infectious** and **Wasting Diseases** there is a deficient secretion of HCl due to constitutional causes, probably nervous in character.

In **Carcinoma** of the stomach there is usually a complete absence of free HCl due to both local and constitutional interference with cell activity.

Excessive Eating may produce temporary anacidity of the gastric contents, on account of the inability of the stomach to secrete a relatively sufficient amount of acid for the quantity of food.

Hyperacidity may be due to increase of HCl or of organic acids, as lactic acid.

In **gastric ulcer** the secretion of HCl is occasionally increased by the hypersensitiveness and irritation of the cells surrounding the ulcer. More often the irritative effect of the hyperacidity causes the ulcer.

In the early stages of **gastritis** (**dyspepsia**) there is hypersecretion of HCl from the same causes.

Excess of **lactic, butyric** and **acetic acids** results from fermentation of carbohydrates. These fermentations occur where there is delay in the stomach due to another impairment. Hence they are found in dilatation, organic obstructions of the pylorus, malignant infiltrations, etc.

Where **lactic acid** is constantly excessive, the **HCl** constantly absent, carcinoma may be usually diagnosed.

Neurotic hypersecretion occurs in neurosis of the stomach, such as neurasthenia, hysteria, and may be constant or periodic. The increased acidity frequently irritates the sensitive sensory nerves of the stomach, causing sensations of hunger and through them the solar plexus, causing gastralgia, both of which are usually relieved by food.

Nervous Dyspepsia is regarded by Leube as a group of cerebral impressions made by the irritative effect of an ordinary digestive process upon hypersensitive nerves of the stomach, and the eructations which occur in this condition as a form of motor reflex from such irritation. Another afferent phenomenon is the hypersecretion of HCl referred to under the preceding section, causing sensations of hunger and gastric pain, relieved by food.

Indigestion, i. e., an abnormal length of time

necessary for stomach reduction of food, may result from eating unpalatable or insipid food, owing to the deficiency of "psychic" secretion in the stomach.

THE INTESTINAL TRACT.

Diarrhea and constipation, representing respectively increase and diminution of intestinal peristalsis, are almost invariably due to a disturbance of the nervous reflex governing this muscular function.

Diarrhea, the expression of an exaggerated peristalsis, is most frequently due to a catarrh, either primary or secondary, which renders the intestinal nerve ends irritable and leads to over-stimulation of the peristaltic reflex.

Psychical Diarrhea, such as accompanies sudden emotions, is doubtless brought about through vasomotor mechanisms. Certain emotions, such as fright, inhibit the splanchnic vaso-constriction, causing a congestion of the splanchnic vessels and over-stimulation of the intestinal reflexes.

Intestinal Dyspepsia is attended with diarrhea, because the undigested food acts as a foreign irritant to the intestinal nerve ends and excites peristalsis. Undigested food is found in the stools. (Lienteria.)

In **typhoid fever** and **cholera** the bacteria and

their toxins focus as a rule in the bowels, producing intestinal catarrh and exciting peristaltic reflex. Intestinal tuberculosis operates the same way.

Gall stones induce diarrhea by mechanical irritation of the intestinal nerves.

Worms excite peristalsis both by mechanical and chemical stimulus.

In **intestinal obstruction** the accumulation of material above the obstruction acts as an abnormally powerful mechanical stimulus and increases peristalsis, but naturally the increased peristalsis does not result in diarrhea. The bowel being greatly distended, the vigorous peristalsis can frequently be seen through the abdominal wall, and in connection with absence of defecation makes a characteristic syndrome, as well as a measure of the extent, of intestinal obstruction.

Exposure to Wet and Cold may produce diarrhea by means of vaso-motor reflex, the skin stimulus causing congestion of the splenchnic vessels and thus overstimulating peristalsis. Burns of the skin frequently act in the same way.

Gastric Indigestion causes diarrhea by reason of the insufficient liquification of the chyme, which therefore exerts too great a mechanical stimulus on the nerves of the intestine.

Reflex Nervous Diarrhea may result from reflex irritation from some other diseased organ.

Constipation, the expression of diminished peristalsis, is frequently due to faulty innervation of the peristaltic reflex, either in its afferent or efferent phase.

Neurasthenia, Hysteria, Anemia, Chlorosis, etc., are attended by constipation because of the diminution in nerve energy, coupled with a debility of the muscles of the intestines.

In **chronic constipation** the nerves lose their peripheral sensibility on account of neglect to respond to the reflex desire for defecation. Later the intestinal muscles become flaccid.

In certain **spinal diseases** such as **myelitis** and **meningitis,** constipation results from involvement of the reflex arcs.

All **fevers** in which there is no specific intestinal catarrh (as in typhoid, vide supra) are attended by constipation. This is due partly to the rapid absorption of moisture from the bowel contents, making their passage difficult, partly in diminution in the amount of contents, lessening the reflex stimulus, and partly to malnutrition of the intestinal muscles, or weakening their intrinsic power.

Diabetes is attended by constipation, due partly to rapid absorption of moisture and

partly to diminution of bowel contents, much of their normal solid constituents being eliminated by the kidneys.

Constitutional Diseases (tuberculosis, syphilis, etc.) unless specifically attacking the bowel with catarrh, exhibit constipation, due to malnutrition of the muscle and faulty innervation.

Fat in the feces, to any abnormal amount, is usually regarded as presumptive evidence of pancreatic disability, but can only bear this diagnostic interpretation in the absence of symptoms pointing to suppression of bile, intestinal tuberculosis, diarrhea, or large ingestions of fat. (See Composition of Feces.)

Lipuria (fat in the urine) is subject to the same provisional interpretation.

LIVER.

The function of the liver is threefold: (1) The secretion of bile, (2) the elaboration of urea, and (3) the formation and storage of glycogen.

Jaundice, due to suppression of bile, is a frequent symptom in parenchymatous diseases of the liver, notably in acute yellow atrophy, hepatic cirrhosis, malignancy, syphilis and abscess.

Uremia, now generally recognized as a suppression of urea rather than its retention, is

undoubtedly the direct result of abnormal hepatic metabolism although the exciting cause is the failure of the kidney to excrete urea. This phase of the hepatic function is too obscure to furnish any definite contribution to functional diagnosis.

Starvation of the tissues, especially the muscular tissues, accompanies almost every degenerative disease of the liver.

Coma due to retention and toxemia of metabolic products also accompanies all severe parenchymatous diseases of the liver, such as yellow atrophy, cirrhosis, etc.

Intestinal Indigestion accompanies suppression or retention of bile, especially of fat-splitting ferment whose activity it increases threefold, and of the proteolytic and amytolytic ferments whose activity it doubles.

These **digestive disturbances** are therefore always seen in conditions which interfere with the elaboration or discharge of bile, as yellow atrophy, malignancy, hepatic abscess, cirrhosis, cholecystitis, gall stone, etc.

Nervous disturbances also attend bile suppression or retention, due to the suspension of the excretory office of the bile and the consequent reabsorption of pigments, cholesterin, licithin, and similar excretory materials.

Retention of Bile results from any condition which (a) interferes with the proper performance of the gall-bladder reflex, or (b) obstructs the passage of the duct, in which case the bile pigments are absorbed and appear in the epithelial tissues as jaundice.

Familiar examples of the first variety are malaria, sepsis, icterus neonatorum, pernicious anemia.

Of the second class, conspicuous instances are gall stones, cholecystitis, malignant growth, and cirrhosis.

Suppression or **Retention of Bile** invariably manifests itself by an absence of these pigments from the feces; resulting in a clay-colored stool, common to all those diseases already enumerated as interfering with flow of bile.

Excessive bile, on the other hand, exhibits an excess of pigments in the feces, producing dark green or very yellow stools, seen principally in excessive proteid feeding and in gall stones following an obstruction of the duct and consequent accumulation of bile. (See Feces.)

Biliousness, as commonly understood by the laity, is due to the poverty of the bile in acid salts. In their comparative absence, fats are very hard to digest or to dispose of, hence the intolerance of the patient to greasy foods; and

the stimulation of bile-flow is weakened, causing a general diminution of bile and bile function.

Innervation: The secretion of bile does not appear to be under the control of any special set of nerves, but is a constant function, dependent for its regulation only upon the vasomotor influences of the hepatic vessels, and probably also on the character of the blood flowing through them.

Discharge of Bile: Normally the bile is not continuously given by the liver directly to the duodenum, but is stored in the gall bladder, guarded by a sphincter, and discharged at intervals during digestion. The ejection of the chyme into the duodenum acts as a stimulus for the reflex which relaxes the sphincter, contracts the gall bladder, and throws the bile into the duodenum. This reflex is mediated by the vagus and splanchnic nerves. Bruns asserts that the stimulus depends on the character of the chyme; acids, alkalies and starches being inert, proteids and fat or their products effective.

Swollen Spleen: The spleen is an organ whose function or functions are very obscure. It is supposed to (1) generate new corpuscles, (2) furnish a graveyard for red blood corpuscles, and (3) assist in the formation of uric acid.

Although a swollen spleen is the accompaniment of many diseases, particularly typhoid fever, malaria, leukemia and secondary anemia, there is not sufficient data as to its physiological function to establish any diagnostic relationship between it and the disease in question.

ELIMINATION.

Albuminuria: In their normal condition the epithelial cells of the kidneys are only permeable to those inorganic and organic constituents of the blood which make up the normal constituency of urine. In diseased conditions of the kidney which disable these cells, however, other elements are let through the glomeruli and tubules, chief among which is albumin. This phenomenon is known as albuminuria, and is common to those diseases which injure the epithelial tissue of the kidneys.

There are two varieties of albumin found in pathological urine, **Nucleo-Albumin** and **Serum Albumin.** Of these the former is derived from the disintegration of the epithelial cells themselves, and is therefore not diagnostic of renal disease, as the disintegrated cells may come from other parts of the urinary tract. Serum albumin, on the other hand, is derived directly from the blood, and is evidence of the disability and permeation of the renal tissues.

The gravest forms of albuminuria, of course, are found in diseases of the kidney proper, i. e., in all forms of nephritis, amyloid disease, tuberculosis, cancer, abscess, calculus, etc.

The next severest occurrences of it are seen in those circulatory diseases, and diseases affecting circulation, which produce a secondary congestive effect upon the renal tissues, as heart diseases, hepatic cirrhosis, tumors, anemia, etc.

All infectious and toxic conditions cause a transitory albuminuria by the extension of their poisonous action to the renal cells.

Hematuria (blood in the urine): Is the result of grosser lesions of the kidney tissue, allowing blood en masse to enter the glomeruli and tubules. (This, of course, assuming that the blood originates in the kidney, and not in some other part of the urinary tract.)

All of the causes above credited with the power of causing albuminuria may also, by affecting grosser injury, give rise to hematuria.

Hemoglobinuria is, of course, always present when hematuria is, but may occur independently in diseases in which there is great destruction of red corpuscles, e. g., anemia, grave infectious diseases, etc.

Glycosuria: Ordinarily, that is to say, under the pressure in which it normally exists in the

blood, the renal tissues are impermeable to sugar. But when it reaches an abnormally high percentage it is excreted in the urine. This occurs in diabetes melitus and is pathognomonic of that disease.

Neurogenous Glycosuria is a temporary excess of sugar in the urine due to nervous shocks, neurasthenia, post-operative conditions, and other disturbances of nervous functions, and must not be confounded with true diabetes. There is no pancreatic disability in these neurogenic forces; they depend entirely on an exaggerated distribution of sugar by the liver—hence increased muscular metabolism will reduce the glycosuria.

Casts are nothing more or less than particles of renal tissue which have acquired the mould of the tubules in passing through them, and in many cases gained a covering of epithelium. They are of numerous variety, depending upon their composition, or more often upon their appearance, such as hyaline, granular, waxy, fatty and blood casts. True epithelial casts, composed entirely of epithelial cells, are rare. Casts, of course, are found only in destructive processes in the renal tissues, and are diagnostic, when found in numbers, of organic kidney lesions.

Absolute **anuria** is rare, but may be caused

by (a) complete destruction or disability of renal tissue, as in violent acute nephritis and in the last stages of organic kidney diseases, or (b) by complete pelvic obstruction, as in nephrosis, calculus, etc.

Oliguria (diminished secretion) results from any conditions injuring or disabling the secreting area and uncompensated by increased renal blood flow, as in malignant diseases, amyloid degeneration, acute nephritis, later stages of chronic nephritis after compensation has failed.

Oliguria, or diminished flow of urine, is induced by all diseases which lessen the blood flow through the kidneys, e. g., myocarditis, valvular diseases (uncompensated), lung diseases, hepatic cirrhosis, which reduces the blood force, and in all diseases of the kidney itself which constrict the renal vessels.

Polyuria (increased secretion) comes from conditions which, while not completely disabling the secreting tissues, render them abnormally permeable to fluids, as in interstitial nephritis, cirrhosis, diabetes, etc.

The quantity of urine secreted is always increased in those conditions which increase the flow of blood through the kidneys, as in cardiac hypertrophy primary (i. e., not due to contracted kidney) which induces high pressure in the renal arteries, and in cystic degeneration

of the kidney and hydro-nephrosis, which dilate the renal vessels.

Increased and decreased secretion, however, may, and frequently do, result from nervous and emotional conditions, which must be explained by their effect upon the renal function through the nerve channels above described. It is worthy of note in this connection that such influences seem to affect only the glomerular portion of the function, since nervous and psychic polyuria consist always in an increase in the water and inorganic salts, with a corresponding drop in density, while the same types of oliguria exhibit simply a diminution in these ingredients with a corresponding rise in density. The organic secretions do not seem to be influenced in such purely nervous cases.

Hysteria, migraine, neurasthenia, epilepsy, delirium tremens are examples of nervous polyuria.

Post-operative shock, melancholia, cerebral disorders, etc., frequently inhibit the renal function, and may even cause death thereby.

The urine is normally a pale amber-colored fluid, slightly acid in reaction when passed, and of about 1.020 specific gravity. Its acidity depends chiefly upon the presence of acid phos-

phates, of which sodium phosphate is the most important, which again, owe their preponderance to the proteids in the diet. The specific gravity is, of course, largely determined by the proportion of solids in solution, of which urea is the chief.

The average composition of normal urine (which, of course, varies under differing diets and physiological conditions) is as follows:

Water (average daily amount).....1500 c.c.
Urea (approximately).....30 grams
Uric acid.....1 gram
Creatin.....1.5 grams
Sulphuric acid-.....4 grams
Phosphoric acid.....3 grams
Inorganic salts.....10 grams
Pigments.....Variable

The specific gravity of the urine is, of course, increased by any influence which (a) increases the amount of solids, or (b) decreases the quantity of water in the secretion.

Glycosuria (sugar in the urine) is by far the most important of the first-class of pathological conditions. In diabetes mellitus, the percentage of sugar ranges from 0.5 to 8 per cent., and the specific gravity varies between 1.035 and 1.040. In fevers the increase of urea raises the density.

Acute and Chronic Parenchymatous Nephritis are conspicuous examples of the latter variety. In these diseases the glomeruli are congested and degenerated, hence the amount of water secreted is lessened and the density of the urine correspondingly increased.

Specific gravity is decreased, on the other hand, by any condition which (a) reduces the amount of solids, or (b) increases the quantity of water secreted.

Interstitial Nephritis (atrophied or contracted kidney) and amyloid kidney are instances of the former variety.

Diabetes insipidus furnishes a typical example of the latter class. In this disease the quantity of watery secretion is largely increased, but not the secretion of organic solids.

Hyperacidity is found in gout, lithiosis, and acute rheumatism, owing to the preponderance of uric acid; in fevers owing to the abundance of urea; and in diabetes because of the presence of acetone.

Alkalinity occurs in cystitis, prostatitis, malignant diseases of urinary tract, paralyses, and any other condition which causes long retention and consequent fermentation of the urine. Profuse hematuria renders the urine alkaline.

Urea is the most important ingredient of normal urine, as it is also the most important

of the nitrogenous excreta, which are practically all excreted in the urine. Urea is by far the largest of the end products of proteid digestion, and the quantity found in the urine practically determines the amount of proteids broken down in the body in a given time, and other forms of nitrogen found in the urine (creatin, ammonia, salts and purin bodies) and those secreted by other channels (milk, sweat) being a negligible quantity.

The precise rationale of its elaboration is unknown. It is assumed that the ammonium compounds resulting from proteid catabolism reach the liver through the portal system, are there converted into urea, and the latter eliminated by the kidneys. Suppression of liver function results in accumulation of ammonium compounds in the blood, and suppression of kidney function in accumulation of urea. There is evidence, however, that urea is formed to a limited extent by other tissues than the liver.

Increase of urea in the urine is the direct outcome of increased metabolism. Hence it is found in fevers and inflammatory diseases, in diabetes, malaria and pernicious anemia.

Decrease of urea, on the other hand, points to abnormal decrease of metabolism, and is seen in chronic nephritis, gout, rheumatism, malignant and constitutional diseases, not so

much as a result of these diseases, but as an accompaniment, due to the same metabolic disturbance as is causing the disease.

Uremia is sooner or later the upshot of a suppression of urea in the urine. Formerly it was thought to be due to the accumulation of urea in the blood (hence the name), but latterly it is agreed that the process of metabolism of which urea is normally the end product, is diverted, and produces abnormal toxins which are not eliminated by the kidneys, but circulate in the blood and poison the nerve centers.

Obstruction: The ureter is not infrequently the seat of obstruction due to impacted calculus, shreds of malignant growths, stricture, spasm, or external pressure from pelvic and abdominal tumors. The obstruction, of course, prevents the normal on-flow of urine and produces stasis in the renal pelvis (**hydronephrosis**), and if bilateral, eventual suppression of urine. Fortunately bilateral troubles of this kind are rare.

Infection to and from the bladder and kidney is occasionally carried by the ureters, causing pyelitis or cystitis, as the case may be. Owing to the normal downward peristalsis, ascending infection has, of course, not so prolific a starting place.

Decomposition and bacterial fermentation occur if from any cause the urine remains too long in the bladder, in which case it becomes alkaline (ammoniacal)and a source of toxic infection to the urinary tract. Such a phenomenon occurs in any condition which delays the voidance of urine, e. g., enlarged prostate, vesical paralysis, malignancy, chronic cystitis, and brain diseases.

Pyuria, pus in the urine, frequently results from suppurative and infectious conditions of the bladder. Epithelium, blood, and even nucleo-albumin may also be derived from broken-down conditions of the bladder-wall as in malignancy, tuberculosis, calculus, cystitis.

Abnormal desire for urination both as to urgency and frequency, are seen in pathological conditions of the bladder and urethra.

Excessive desire, urgent and frequent, results from conditions which either (a) rapidly distend the bladder to its physiological limit, or (b) render the nerves so sensitive that an abnormally small distension stimulates the reflex.

The former class includes all those diseases which increase the secretion of urine, notably diabetes, nephritis, cardiac hypertrophy, and neuroses.

Of the latter class the chief conditions are those of cystitis and urethritis, in which the inflamed state of the membrane renders the nerves amenable to very slight stimulus. In these conditions a small degree of bladder distension excites the reflex contraction, squeezing a drop of urine into the highly sensitive inflamed urethra, and producing an overwhelming desire to micturate. Small quantities are of course passed at each act.

Over-concentration of the urine, hyperacidity, and other chemical conditions of the urine may also cause increased desire by unduly irritating the afferent nerves. This frequently occurs in fevers, gout, rheumatism, etc. It is probable, however, that these properly belong under the heading of cystitis and urethritis.

Subnormal desire, on the other hand, results from any conditions which (a) diminish urinary secretions, or (b) dull the sensibilities of bladder or urethra.

In the first variety come all those diseases which diminish renal activity, notably chronic nephritis, myocarditis, nephrosis and neuroses.

In the second list are classed malignant diseases of bladder and urethra, tuberculosis, and chronic cystitis and urethritis, in which the tissues are rendered lax and dull; and central nervous lesions which depress the general nervous

activity. In any of these conditions an abnormally powerful distension is necessary to stimulate the reflex.

Involuntary Micturition: Paralysis of the nervous tracts concerned in micturition, either by destruction of the spinal centre (most frequent) or by interruption of the afferent or efferent paths, produces paralysis of the act. In such cases the urine is not voided until distension becomes so great that it is forced out of the bladder by sheer mechanical pressure, after which it dribbles away involuntarily. It is rather an uncommon phenomenon, occurring chiefly in severe forms of spinal disease, e. g., myelitis, sclerosis, late tabes dorsalis, and injuries. It must be distinguished from involuntary micturition, which depends upon a suspension or interruption of the inhibitory influence of the brain. In this condition the urine is voided involuntarily, as soon as distension is sufficient to stimulate the bladder reflex, the bladder being completely and as a rule convulsively emptied at each orgasm. This phenomenon occurs in cerebral diseases, profound coma, and certain neuroses. Abnormally deep sleep occasionally induces it (enuresis).

Psychic conditions may, by direct operation through the vesico-spinal centre, influence the act of micturition, either by suspending inhibi-

tion and precipitating the reflex (most frequent) or by inhibiting the reflex itself. Conspicuous instances of both these phenomena are frequently met with in cases of psychic shock, hysteria, and melancholia.

Abnormal desire for stool, both as to frequency and urgency, is seen in pathological conditions of the rectum and anus.

Excessive desire results from any conditions which (a) rapidly distend the bowel to stimulation point, (b) render the rectum unduly irritable so that an abnormally small distension (or even no distension at all) stimulates the reflex.

In **diarrhea** and **dysentery** both of these factors are usually active in producing frequent and urgent desire for stool. It must not be forgotten, also, that the liquid character of the feces makes a much more vigorous contraction of the sphincter necessary in order to keep them inside the rectum.

In **proctitis** the inflamed condition renders the rectum so sensitive to stimulus that the presence of small quantities of feces induces the reflex act of defecation with great urgency and pain (tenesmus).

Subnormal desire arises from any condition which (a) reduces the quantity of feces reach-

ing the rectum in a given time, or (b) renders the return abnormally irresponsive to stimuli.

Constipation (diminished peristalsis) from any cause, of course, comes under the first head, while under the second may be classed malignant diseases of the rectum, tuberculosis, chronic proctitis, which render the rectal walls lax and dull, and central nervous lesions which depress general nervous activity. In any of these conditions, considerable distension is necessary to stimulate the reflex, and in many of them the pain attending defecation induces a voluntary retention.

Involuntary Defecation results from an interruption of the inhibiting influence of the higher centres upon the sphincter. As soon as the distension of the rectum is sufficient to stimulate the reflex the feces are involuntarily voided by a vigorous contraction of the muscles concerned. This symptom occurs in cerebral diseases, profound coma, and certain neuroses and very exceptionally in deep sleep. It must be differentiated from

Rectal Paralysis, depending upon destruction of some part of the reflex tract concerned in the act of defecation. In these cases there is no defecation until the rectum becomes so full that the feces are forced out by physical pressure, slowly and with no effort. This is

seen in severe forms of spinal disease as in myelitis, sclerosis, late tabes dorsalis, and injuries involving the lumbar region, and rarely in neuritis by implication of the peripheral neuron concerned in the act.

Psychic Influences may, by direct operation through the anal centre, modify the act of defecation either by suspending inhibition of the sphincter and inducing involuntary defecation (most frequent), or by inhibiting the reflex itself and retaining the feces. Psychic shock, hysteria, and neurasthenia furnish instances of both kinds.

Feces: Very little absorption of anything but water takes place in the large intestine, and the alkaline reaction in this part of the tract favors bacterial putrefaction. By the time, therefore, the material reaches the rectum, under normal conditions, three consummations are reached: (a) the substance remaining contains only waste matter; (b) it has acquired a relatively solid consistency, and (c) it is in an advanced stage of decomposition. It is then known as the feces, and is then voided per anum.

The quantity of feces varies with the amount and nature of the food ingested, the average quantity being from 140 to 200 grams in twenty-four hours.

Abnormal Consistency: Aside from the variations due to diet, the consistency of the feces varies directly with the length of time they remain in the large bowel. Under vigorous peristalsis (diarrhea)they pass very rapidly, there is no time for any great absorption of water, and the stools are usually liquid. In diminished peristalsis (constipation), on the other hand, they remain in the bowel a long time, are inordinately drained of fluid, and are therefore dry and hard.

The temperature of the feces, owing to fermentation, is higher than the body temperature—hence in taking rectal temperature, care should be exercised not to insert thermometer in a mass of feces.

Composition: The constituents of the feces, of course, vary with the diet and other circumstances. Generally speaking, however, they contain the following ingredients:

Undigested foodstuffs (principally fats).

Products of intestinal secretions (nitrogen).

Products of bacterial putrefaction (principally indol and skatol).

Bile salts and pigments (uro and starcobilin).

Inorganic salts.

The characteristic color of the feces is due to the bile-pigments; their odor to the skatol.

Lienteria, i. e., an abnormal quantity of undigested food in the feces, indicates, of course, that the alimentary tract is not properly disposing of the food ingested. It occurs in all digestive disorders. The particular stage of indigestion in which these lienteria are found (chemically) may furnish information as to the precise part of the alimentary process at fault.

Mucus in the feces is indicative of catarrh when found in large amounts.

Clay-colored stools signify a lack of bile pigments and indicate a suppression or obstruction of the gall bladder or common duct, as in jaundice, cholecystitis, gall-stones, hepatic cancer, etc.

Blood may come from any part of the intestinal tract and from many causes. Hemorrhoids are the commonest cause. Enteritis, ulceration, cancer, tuberculosis, are less frequent causes. In these cases the blood is usually comparatively bright red. Black blood (altered blood) originates in the upper alimentary tract, as from duodenal, or gastric ulcer, swallowed blood, hepatic cirrhosis, etc., and is partially digested during its passage.

Gall stones are often found in the feces. Their significance is obvious.

Microscopical and Chemical Contents: Latterly Nothnagel, and still later Adolph Schmidt, have inaugurated a more thorough functional diagnostic of digestive and intestinal disorders by means of a minute and systematic examination of the feces, similar to the methods employed in gastric functional diagnosis. The system is, however, as yet rather too imperfect to enter into a practical text book.

Increased fat in the stools indicates (a) a deficient secretion or flow of bile, as in jaundice, cholecystitis, hepatic cancer, etc., (b) disturbance of pancreatic secretion, hindering fat digestion, as in pancreatitis, or (c) intestinal abnormalities interfering with absorption, as in intestinal tuberculosis, malignancy, amyloid disease, etc.

THE NERVOUS SYSTEM.

Increased Nervous Irritability: The irritability of both cells and axon is greatly modified by the action of certain drugs, e. g., strychnine, ergot, belladonna and phosphorus.

Delayed Conduction: Pathological conditions of definite tracts and environs frequently increase the time of the passage of an impulse (delayed conduction). A conspicuous example of this is seen in peripheral neuritis (inflammation of the lower sensory axons).

Certain drugs depress these functions, notably opium, chloral, Curare, bromides and Indian hemp.

Sensory Paralysis, or Anesthesia, results from interruption in the course of these lower sensory neurons, or injury to the peripheral endings, which paralysis is, of course, limited to the area supplied by the neurons involved. Such anesthesias are due to (1) traumatic injury to nerve-ends as from burns, skin diseases, etc., (2) vaso-motor disturbances at the peripheries, as anemia or congestion, and (3) lesions along the course of the axons, from tumors, inflammation, etc. Peripheral and multiple neuritis are conspicuous examples of this latter class.

True sensory paralysis is rare—complete anesthesia still rarer. When it does occur it is mostly peripheral in origin, as already described.

Given the **integrity of the sensory peripheries,** however, it is rarely that the sensory impulses cannot find some path or other by which to reach the brain. Interruption of any of the ascending tracts of the cord never produces more than partial paralysis, except in rare cases or growths or degeneration which involve the whole section of the cord, in which case, of course, both motor and sensory paraly-

sis are complete. Spinal anesthesias are usually bilateral.

Anesthesias due to **cerebral lesions, hemorrhage, tumors,** softening, etc., are extremely rare, difficult to demonstrate, and never intense. They are usually unilateral.

Syringomelia is the disease which affords the most interesting manifestations of sensory conduction. The anatomic basis of this disease is the formation of cavities in the substance of the cord, and it is clinically attended by some very peculiar and characteristic sensory phenomena, chief among which is a discriminate insensibility to the various stimuli of temperature, pressure and touch (dissociation of sensations).

A far more common sensory phenomenon is that of irritation, due to primary inflammation of either the lower or upper sensory neurons, and manifested clinically by pains referred to the periphery of the affected neuron. These pains are seen in locomotor ataxia (degeneration of the posterior column), multiple neuritis (inflammation of lower neurons), myelitis (inflammation of the spinal tracts), and other similar diseases, in their early stages; on supervention of degeneration or compensation they give way to partial anesthesia.

Motor Paralysis results from interruption in

the course of these lower efferent neurons, or injury to their peripheral endings, limited to the area supplied by the interrupted neurons. Such paralyses are due to (1) traumatic injury to nerve ends, as from burns, skin diseases, etc., (2) lesions in the course of the axons, as tumors, inflammations, etc.

Owing to the fact that the lower motor and sensory neurons travel in the same sheath, lesions of these lower neurons almost always involve both, and hence produce motor and sensory paralysis. A double paralysis of a limited area is therefore suggestive of a lesion of the lower neuron.

Any interruption in the course of the spinal tracts produces motor paralysis in the part below the interruption. A careful examination of the muscles involved in such paralysis should therefore enable the diagnostician to locate the level of the lesion; and so it usually does, to a certain extent, but unfortunately not with anything like the definiteness that the data would lead one to expect. In fact, actual spinal paralyses are not nearly so sharply defined as hysterical paralyses.

Poliomyelitis (inflammation of the anterior horns) progressive muscular atrophy, which later attacks the anterior root cells, and amyotrophic lateral sclerosis (involving the crossed

pyramidal tracts) all produce motor paralysis by interrupting the motor tracts of the cord. In multiple sclerosis, there is seldom true motor paralysis, because in spite of the diffuse nodules, the neuraxons usually persist and functionate.

Myelitis, which influences all the tracts, produces motor paresis.

Crossed Paralysis: Injury to a motor tract above the point of decussation will produce a crossed paralysis, i. e., a paralysis of the opposite side of the body to the lesion, but on the same side of the head and neck, as the cranial nerves are given off before decussation takes place. In case the cranial nerves are not injured (a rare combination) there is no way of diagnosing the crossed nature of the paralysis.

Injury to a motor tract in the lateral column of the cord will produce a paralysis on the same side below the seat of the lesion.

Injury to the direct motor tract will give paralysis on both sides. This class of paralysis is frequently associated with lesions of the lateral sensory tract, involving ataxia.

As a matter of practice, however, paralyses due to lesions in the spinal tract are almost invariably bilateral (paraplegia) because the pathological process usually involves both sides of the cord.

Atrophy: In diseases of the nerves where the lesion is situated between the root-cells and the periphery, or in which the root cells themselves are involved, there is rapid atrophy of the muscles concerned in addition to paralysis. Conversely, where rapid atrophy is associated with paresis, the lesion may be safely located in one of these positions.

In **poliomyelitis** there is atrophy because the anterior horns are themselves inflamed and later degenerated.

In **diffuse myelitis** the same is true.

In **progressive muscular atrophy** the degeneration begins in the lower neuron, often in the muscles, hence atrophy precedes paresis.

In **lateral amyotrophic sclerosis** the degeneration begins in the spinal tracts and later attacks the root cells, hence paresis precedes atrophy.

In **multiple neuritis** the lower neurons are inflamed, hence there is atrophy if the inflammation continues for any length of time.

In **diseases of the cord which involve only the tracts,** and in purely **cerebral diseases,** there is no such atrophy, because there is no interruption of the course between the limb and the root-cell, the lesion being above the root-cell.

Incoordination: Anything which interrupts the passage of muscle sense impulses deprives the cerebellum of information which is essential to the performances of its co-ordinating function, and results in incoordination, manifested clinically by inability to accomplish purposeful movements of the parts involved, staggering gait, loss of balance, inability to perform delicate tasks with hands, etc.

Conversely, these clinical symptoms always point to interruption of muscle sense impulses. Whether the interruption is in the lower or upper neuron must be determined by other symptoms.

In **myelitis** the spinal tracts, and in **neuritis** the peripheral sensory neurons, are inflamed, and cannot transmit impulses; hence ataxia results.

In locomotor ataxia the posterior column (Goll and Burdoch) are degenerated; hence the muscle sense can no longer be transmitted along these tracts and ataxia results.

Lateral Scleroses, by virtue of the degeneration of the lateral tracts (Flechsig and Gowers) produce a similar result.

Absence of a reflex may indicate an interruption in either of the lower neurons in the afferent or efferent, or a destruction of the

spinal arc in the cord. The former cause may be excluded by absence of other indications, such as flabbiness and atrophy of muscles. Far the greater proportion of reflex failures are due to injury to the spinal arc.

Naturally the reflexes whose arcs are lowest down in the cord are the most commonly utilized in diagnosis, as they indicate the condition of the cord above the arc. Of these low-down reflexes, the **knee-jerk** (Patellar) and **tendon Achilles** are the most easily elicited and constant.

The **normal knee-jerk** indicates integrity of both afferent and efferent (lower) neurons of the third lumbar nerve, of the commisural arc at the third lumbar segment, and of the tracts from the third lumbar segment up to the grain. Thus a normal knee-jerk practically assures integrity of the entire spinal tract.

Exaggeration of the knee-jerk indicates an interruption in one or more of the descending spinal tracts between the third lumbar segment and the brain, cutting off the inhibitory influence of the brain. For the same reason an exaggerated reflex is associated with **hypertonus of the muscles.** This condition is seen in multiple sclerosis, where the tracts are studded with nodules, amytrophic lateral sclerosis, in which the lateral motor tracts are degen-

erated (very marked), in cerebellar disease, where the coordinating influence upon the tendons is cut off, and in all cerebral lesions. Central myelitis gives this symptom, because it involves the upper and not the lower neurons.

Absence of Diminution of the knee-jerk indicates interruption in the afferent or efferent neuron of the third lumbar nerve, or injury to the commissural arc, or disease of the posterior tract of the cord which includes destruction of the arc. The knee-jerk is abolished or lessened in neuritis and progressive muscular atrophy because of the inflammation of the lower neurons; in locomotor ataxia because of degeneration of the spinal arc. In the latter disease a careful examination of the various reflexes whose arcs are at different levels will indicate the upward limit to which the degenerative process extends. In diffuse myelitis the same occurs because the roots of the lower neurons are involved.

Both **exaggeration** and **diminution** of reflexes may be due to exalted or depressed conditions of the cord, caused by drugs or mental emotions, but these indications are as a rule easily recognized or excluded.

The **tendo Achilles** and **plantar reflexes,** of course, give information concerning the spinal tracts, from a still lower level, and these and

other reflexes exhibit certain peculiarities and give information concerning their own specific neurons.

Differentiation of Spinal and Cerebral Paralysis: Interruption of the course between the brain and the spinal neuron, in other words, injury to or division of the upper neuron, interferes with the inhibitory influence of the brain upon the tonic function of the cord, and an excess of tonic energy is poured out by the latter into the muscles. This is manifested clinically by a spastic condition of the muscles involved, and an exaggeration of reflex.

Interruption of the course between cord and periphery, i. e., injury to or division of the lower neuron, results in a cutting off of the tonic current, manifested clinically by a flaccid atonic condition of muscles involved and absence of reflex.

Conversely, therefore, spastic paralyses and exaggerated reflex indicate injury to the upper neuron or brain. Flaccid paralyses and absence of reflex point to injury to the lower neuron. This should be one of the first diagnostic points to be determined in the investigation of a paralytic condition.

In **peripheral neuritis** (inflammation of the lower neuraxon), poliomyelitis (inflammation of anterior roots), and progressive muscular

atrophy (degeneration of the lower neuron), the tonal path is interrupted, and these diseases are characterized by flaccidity of the muscles involved, and diminished reflexes.

In **lateral amyotrophic sclerosis,** there is first spasticity and exaggerated reflexes, as long as the process is confined to the lateral tracts. followed by flaccidity and diminished reflexes when the root cells are attacked.

In **multiple sclerosis,** involving only the spinal tracts, and in all cerebral paralyses, the muscles are spastic, and the reflexes are exaggerated.

In **central myelitis** there is spasticity, because the upper neurons only are involved; in **diffuse myelitis** the reverse, because the roots of the lower neurons are inflamed.

Spasticity manifests itself in complete paralyzed limbs by resistance to passive motion. In partially paralyzed limbs it is shown by a jerky character of the movements; in the legs by a peculiar dragging walk (spastic gait).

Bulbar Paralysis: Injury to, or disease of, the medulla, if it involves, as it usually does, the respiratory center, of course, paralyzes respiration and causes death. Dislocation of the atlas as a rule produces instant death by sudden trauma and compression of the medulla.

In **amyotrophic lateral sclerosis** the atrophy eventually reached the medulla and ends the scene of paralyzing either respiration or deglutition. Multiple neuritis frequently has the same outcome from inflammatory investment of the lower neurons concerned in respiration.

Inco-ordination: Interruption of the passage of sensory impulses from any of these sources to the cerebellum, or incapacity on the part of the cerebellum to receive them, produces inco-ordination, manifested clinically by impairment of purposeful movements of the parts involved. The interruption may occur either in the lower or the upper neuron, or the cerebellum itself may be the seat of the lesion. Its location must be determined by considering accompanying symptoms.

Peripheral neuritis produces ataxia by interrupting the transmission of impulses from the skin and muscles at the periphery.

Locomotor ataxia manifests ataxia because of injury to the posterior columns of the cord —one of the paths of the upper sensory neurons (vide supra).

In **multiple sclerosis** the nodules are scattered through the tracts, effecting a partial and diffuse interruption of sensory impulses, the result being a partial form of ataxia which is now understood to be the explanation of intention tremor.

In **lateral amyotrophic sclerosis,** on the contrary, the process is always sharply limited and the cerebellar tract left intact; hence, amid all the havoc wrought by this formidable disease, coordination is unimpaired.

Myelitis, in which all the tracts are involved, gives ataxia, but the motor paralysis masks it.

In a **hereditary** form of **locomotor ataxia,** known as Friedreich's Disease, the inco-ordination extends to the muscles of speech, and the muscles of the trunk are involved, making it difficult to maintain static as well as dynamic equilibrium.

That the **sense of vision** plays an important part in coordination is proven by the fact that in all the ataxia diseases referred to, the incoordination becomes more marked when the eyes are closed (Romberg's symptom).

Cerebral Motor Paralysis: Trauma or disease of the great sensory and motor tracts during their passage through the capsule and pons, where the fibres are compressed into a very small sectional area, naturally includes all of the neurons in the injury, and produces a paralysis of all the parts supplied by the tract—partial or complete, according to the extent of the injury or compression. If the lesion is above the cranial nerve roots (as it usually is), the result is a crossed paralysis involving

the same side of the head and neck and the opposite side of the body. If it is below the cranial nerve nuclei, the paralysis involves the opposite side of the body.

Hemorrhages into the capsule are very common, as are also embolisms of the middle cerebral artery, tumors, gummata and abscesses involving the capsule. The first two lesions usually cause a sudden and complete paralysis; the other three lesions usually produce gradually progressive hemiplegia.

Cerebral Localization: Experiment and observation have demonstrated that certain areas of the cortex, being stimulated, functionate various parts of the body; one area, for example, always moving the leg, another the arm; and still smaller points are found in these areas which influence detailed sections of the parts in question, as the foot and toes, hands and fingers. The same is true of special functions, such as speech, hearing, etc. Hence we conclude that every part of the body is represented, afferently and efferently, by a certain definite area in the cortex, although the sensory areas, naturally, cannot be mapped out with the definiteness of the motor areas.

Monoplegias: Inasmuch as the motor centres of the cortex are separated from each other by a more or less measurable distance,

traumata and degenerative processes of the cortex, unless extraordinarily extensive, do not involve more than one or two of these centres. Hence the paralyses produced by cortical lesions do not, as a rule, involve more than one group of muscles, and are known as **monoplegias.** Conversely, a monoplegia may, as a rule, be diagnosed as due to a cortical lesion, for in no other part of the cerebro-spinal course are peripheral groups represented by neurons sufficiently isolated to incur separate injury.

In certain types of **cortical epilepsy** the central irritation starts in one of these cortical centres, and the fit begins with a corresponding disturbance in the group of muscles concontroled by that centre (Jacksonian Epilepsy).

Location of Motor Areas: The motor areas are for the most part situated along the fissures of Rolando and Sylvius, and a description of the probable location of the centres for the most important parts of the body will be found in the accompanying table. The location of special senses centres will be indicated in discussing those senses.

It will be readily seen from the table and illustration that the arrangement of the centres facilitates the occurrence of monoplegias. These are quite frequent in practice and it is

usually a simple matter to diagnose the exact spot in the brain at which the lesion exists. Frequently, too, the lesion will include two adjoining centres, such as those of the face and arm, or of the arm and leg, and a paralysis of these two regions is then produced, which is called an **associated plegia.** But it is apparent that an associated cortical paralysis of the leg and face, without paralysis of the arm, cannot occur as the result of a single cortical lesion.

The greatest care must be exercised in differentiating between the various centres concerned in the functions of speech, hearing and writing, and the effect of injury to these foci. Each of these functions is made up of two distinct operations of the brain, performed by two distinct and separate centres, which may be classified as follows:

Hearing: (1) Perception of sound; (2) storing of sounds (auditory memory).

Sight: (1) Perception of images; (2) storing of images (visual memory).

Speech: (1) Recognition of stored sounds (spoken words); (2) motor speech.

Writing: (1) Recognition of stored images (written words); (2) motor writing.

It will be readily apparent that the function of speech depends upon that of hearing since

sounds cannot be reproduced which have not first been perceived and memorized. In like manner the function of writing depends upon that of sight, since the images cannot be reproduced which have not been first perceived and memorized. Psychologically, the acts of storing and reproducing an impression are identical ("there can be no impression apart from expression").

It will be further seen that the centres for these intradependent functions are situated immediately above each other in the brain as follows:

Motor speech,
Memory of Spoken Words,
Hearing,
Arm and Hand,
Memory of Written Words,
Vision.

Pure Deafness: A lesion of the auditory centre or centre of pure hearing, produces inability to perceive sound. The current conveyed by the auditory nerve produces no effect upon it. This lesion is not necessarily fatal to speech, in these days of modern resource and patience, as the patient may be taught by sight, and, in the absence of sight, even by thought, to recognize and reproduce motions of the mouth and tongue. This form of deafness is called pure deafness.

Word Deafness: A lesion of the centre of sound memory produces inability to store sounds, and therefore to recall them. This form of deafness is called word deafness, and is fatal to intelligent speech, because the word desired to be spoken cannot be thought of. In regard to speech this is known as **amnesic aphasia.**

Pure Blindness: A lesion of the visual centre produces inability to perceive an image. The current conveyed by the optic nerve makes no impression upon it. This form of blindness is called pure blindness and can be overcome by patient education so far as the ability to write is concerned.

Word Blindness: A lesion of the visual memory centre produces inability to store and hence to recall images. This form of blindness is called word blindness, and is fatal to intelligent writing because the word desired to be written cannot be thought of. In regard to writing this is called **amnesic agraphia.**

Aphasia: The form of aphasia dependent upon injury to the sound memory centre (amnesic aphasia) has already been described. The other form of aphasia is that which results from injury to the motor mechanism of speech and is called **motor** or **pure agraphia.**

Agraphia: The form of agraphia resulting from injury to the centre of visual memory (amnesic agraphia) has been described. That form which comes of injury to the motor mechanism of the arm and hand is called **motor** or **pure agraphia.**

Soul Blindness and Deafness: There is still another form of word blindness and deafness in which sounds are heard and images seen, and both are stored and reproduced, but no connection is perceived between the sound or the image and any conception of the mind. This is known as soul blindness and deafness. It depends on no special centre, but upon a general dissociation of the cerebral areas.

TECHNIQUE OF COMMON NEUROLOGICAL TESTS.

Light Reflex. Throw a bright light suddenly into each pupil separately, the other eye being meanwhile excluded from vision. If the reflex is present, and normal, and pupil will quickly contract. If it be absent, no change will occur. If it be diminished, the reaction will be sluggish and incomplete. Then, with both eyes open, throw the light into one pupil and watch the response of the other (consensual reflex).

Sources of error: Be careful to have the accommodation relaxed during the test, or its

value is annulled; for if the accommodation is in play, we cannot tell whether the contraction of the pupil is due to accommodation or to light reflex. Also be sure there are no adhesions of the iris, as these, of course, mechanically prevent contraction of the pupil.

Wernicke Pupillary Reaction. With the patient looking straight in front of him, throw a narrow beam of light from a converging lens on to the blind side of the retina from the opposite visual field. If the reflex is present, both pupils will contract; if not, no reaction will occur.

Source of error: Be careful not to allow the beam of light to fall on the sound side of the retina. This can be avoided by throwing the light from the extreme opposite limit of the visual field, so that it strikes the eye at a very slanting angle.

Accommodation Reflex. Have the patient fix his eyes upon a small object (e. g., a pencil, or the operator's finger) held several feet away. Gradually bring the object nearer to the patient's face, instructing him to keep his eyes fixed upon it. If the reflex be present, the pupils contract as the object approaches them. If not, no change in the pupils occurs.

Source of error: Be sure there are no ad-

hesions of the iris, as these will mechanically prevent the pupil from contracting.

Paralytic Strabismus: Have the patient follow a small object (e. g., a pencil, or the physician's finger) as is gradually brought nearer to the eyes, as described above; and also follow the movements of the same object from side to side. If the strabismus be concomitant (i. e., the result merely of error of refraction) the defect in the muscle will be manifested only in the first of these two tests (convergence); in the second (conjugate movement) the muscles will all function normally. If, however, the strabismus be paralytic, the muscle defect will show in both tests.

Nystagmus: Have the patient follow with his eyes the lateral movements of an object, to and fro, a few feet from his eyes. If nystagmus be present, the eyes will oscillate laterally under this test, and vertigo quickly supervene.

Tendon Reflexes: All tendon reflexes are to be elicited on the same principles of technique. Have the limb, or portion of the limb, to be tested, hang loosely from the nearest proximal joint (e. g., the forearm from the elbow, the hand from the wrist, etc.) with the muscles wholly relaxed and the tendon on the stretch. Now sharply tap the tendon with a heavy percussor. The normal response is a

moderate contraction of the extensor muscles, giving the dependent part a slight, quick jerk. If the reflex be exaggerated, the jerk will be correspondingly convulsive and high, but not necessarily quick. If absent, or diminished, there will be no perceptible response.

N. B.—Since the vigor of tendon reflexes differs in different individuals, the only trustworthy basis for determining exaggeration or diminution of any reflex is a comparison with the same reflex on the opposite side of the body, or with other tendon reflexes in the same individual.

Knee Jerk: Depend the leg loosely from the knee, as described above, and tap the patellar tendon sharply with a heavy percussor. The response is a forward kick of the leg and foot, varying as described under Tendon Reflexes.

N. B.—The common practice of having the patient cross the leg to be tested over the other is poor technique, because the act of crossing the legs brings repression into play. Much the better way is to have the patient sit on a hard table, with the legs just hanging over the edge from the knees. The pressure of the table on the under part of the thigh thus insures relaxation of the leg muscles.

Reinforcement: In children and in self-conscious patients it is sometimes necessary to

employ some positive means of preventing inhibition of the reflex. This is called reinforcement, and is usually accomplished by having the patient hook his two hands together and pull hard on them. Such reinforcement, however, is not of the best, as it is apt to annul the value of the test. When reinforcement is necessary, a psychic form is much preferable, such as distracting the patient's attention by conversation on the part of an assistant while the physician suddenly makes the test.

Babinski Phenomenon: Firmly stroke the outer side of the bare foot with a blunt instrument, such as the handle of a percussor. If the reflexes be normal, the great toe will flex itself, and perhaps the other toes also. If the Babinski phenomenon be present, the great toe will extend itself, and in many cases the rest of the toes will spread themselves out like a fan. The behavior of the great toe, however, is the significant factor.

Source of error: Frequently the great toe, after extending, will reverse itself and flex, as the patient acquires conscious control. Close watch must be kept for the first action of the great toe, as this is the crux of the test.

N. B.—The Babinski phenomenon is normal in infants and in young children.

Ankle Clonus: With the patient lying down

in a thoroughly relaxed state, with one hand grasp the calf of his leg firmly, so as to relax the leg muscles, and raise the foot and ankle. With the other hand grasp the patient's foot at the bottom, and, first flexing it, next suddenly and forcibly extend it toward the shin, and then as suddenly release it altogether. If ankle clonus be present, the calf muscles will be thrown into rhythmic contraction, which may show itself in visible oscillations of the foot, or perhaps is only to be felt by the hand which grasps the calf.

Cremasteric Reflex: Gently stroke the inside of the thigh with a blunt instrument, such as the handle of a percussor. If the reflex be present, the skin of the scrotum on the same side will retract.

Abdominal Reflex: Gently stroke with a blunt instrument the skin of the abdomen just to one side of the umbilicus. If the reflex be present, the skin on the other side of the umbilicus will retract.

Romberg's Sign: Have the patient stand erect, his feet together, his arms close to his side, and close his eyes. If ataxia be present, he will noticeably sway from side to side, and perhaps even fall, unless prevented.

Other Tests for Ataxia. Ask the patient to lift a brimming cup or glass of water to his

lips. If ataxic, he cannot do so without spilling it.

Have the patient try to touch the tip of his own nose in rapid succession with his index finger. If ataxic, he will miss it.

Have the patient try to walk, heel and toe, a straight-marked line, and then turn sharply around. If ataxic, he cannot keep to the line, and upon turning quickly will sway and perhaps fall.

Instruct the patient, either standing or lying down, to trace a circle, or other pattern with his foot or hand. If ataxic, he is unable to do so. This test is especially useful in suspected local ataxias, particularly of the lower limb.

Skull Percussion: Combined percussion and auscultation. Place the bell of the stethoscope firmly on the skull, at about the centre of the vertex, and with a rubber hammer percuss lightly upon symmetrical points of the skull on each lateral side of the median line for comparison. The normal percussion note resembles the "plunking" of a ripe melon, and is the same all over the skull. A thickened skull (e. g., chronic subdural meningitis), or a solid mass immediately under the skull (e. g., a cortical tumor), will give a high-pitched or flattened note over the affected area.

REACTION OF DEGENERATION.

Reaction of degeneration: A similar process of degeneration occurs in a neuron which is cut off from its cell by pathological lesions in the intravening axon, such as new growths, sclerosis, inflammatory congestions, etc. The process is then known as **secondary degeneration,** ascending or descending as the case may be, and is clinically detected and measured by certain abnormal ways in which the neuron and the muscles it supplies react to electrical currents. This is known as the Reaction of Degeneration, and may be briefly stated as follows:

In health, both nerve and muscle react to the faradic current by contracting sharply upon application of the needle, and to the galvanic current by a contraction at the closing and opening of the circuit, but not during the passage of the current, the **Kathode** contraction being more vigorous than the **anode.**

In **complete reaction of degeneration** neither nerve nor muscle reacts to faradic currents; the nerve does not react to galvanic; the muscle contracts slowly and undulatingly to the galvanic, and exhibits as good a reaction to the kathode as to the anode.

Incomplete reaction of degeneration, the re-

actions exhibit intermediate between normal and complete degeneration.

Reaction of degeneration enables us to locate the lesion in the lower neuron or in the root-cells, i. e., between the periphery and the trophic ganglia; and the persistence or improvement of the abnormal reaction indicates a grave or favorable prognosis of the restitution of the integrity of the neuron.

Reaction of degeneration is generally associated with muscular atrophy, because the trophic influence of the ganglion is also exerted upon the nutrition of the muscle.

It does not occur in diseases involving only the cord tract or upper neurons, or in cerebral lesion. Multiple neuritis, involving inflammation of the lower neuraxons; poliomyelitis, inflammation of the anterior horns; and progressive muscular atrophy, involving both lower neuraxons and anterior roots; all give reaction of degeneration. Amyotrophic lateral sclerosis gives reaction of degeneration in its later stages, when it attacks the motor roots.

HEADACHES.

Headaches are the expression, through the nerves of the head, of such numerous and varied body-conditions that it is impossible in such a limited work as this to do more than sug-

gest a very general indication of their significance.

Headaches, as commonly met with in practice, are due to (1) neuro-muscular troubles of the head itself, as seen in **errors of refraction, anomalies of the ocular muscles, indurations of the muscles of the scalp** (rheumatoid headache) and the like; (2) pressure and irritation in and about the skull, as in **diseases of the nose and sinuses**; (3) vascular disturbances, which produce headache, we must believe, chiefly by interfering with the proper nourishment of the brain and the cranial nerves, such as **arteriosclerosis, low blood pressure, anemia,** and certain **vaso-motor spasms** which temporarily constrict the vessels (migraine); (4) toxemias, which poison the central nervous system, in which class are the headaches which attend both **acute** and **chronic infections** of all kinds; and (5) lesions of the Central Nervous System, such as **meningitis, brain tumors and abscesses, cerebral syphilis,** etc., which are always characterized by their extreme severity and their unamenableness to all ordinary treatment.

To the above must, perhaps, be added, as a separate class, the headaches due to **digestive disorders,** in which it is hard to say whether the cause is toxemia or nerve irritation—probably both.

The differentiation of these various types of headache must be arrived at by a consideration of other accompanying symptoms, both subjective and objective.

SPECIAL FUNCTIONS.

Vision.

Unilateral Blindness must be due, in every case, to a lesion in front of the optic chiasm, as only there is the entire visual field of one eye alone represented. Such blindness—which may be partial or complete, may be due either to (1) a disease of the eye itself, e. g., cataract, cyclitis, glaucoma, choroiditis, degenerations of the choroid or retina, arteriosclerosis, tumors of the eye, etc., or (2) lesions of the optic tract anterior to the chiasm, such as optic atrophy, retro-bulbar optic neuritis, tumors of the optic tract, etc.

Bilateral Blindness may be due either to (1) diseases of the eye mentioned above, where both eyes are affected, or (2) lesions of the nervous optic tract in the left frontal convolution of the cortex or at the chiasm, since these are the only two places in the tract where all the areas of both retinae have nervous representation.

Lesions of the optic nerve alone, naturally, affect only that eye which is supplied by the

pathologic nerve. Strictly speaking, therefore, they produce unilateral blindness. Inasmuch, however, as optic neuritis and atrophy are usually due to brain lesions or to systemic disease, both optic nerves are as a rule affected, as that in fact we have bilateral blindness.

Half-blindness of the two retinae (hemianopsia) is due to a lesion of the chiasm, or of the occipital lobe of the brain, since here alone are the respective halves of the two retinae represented by nerve fibres.

Hemonymous hemianopsia, i. e., half-blindness of two similar halves (right or left) of the retinae, is due to a lesion of the occipital lobe, usually a tumor or a gumma.

Heteronymous hemianopsia, i. e., half blindness of dissimilar halves of the two retinae, the left side of one and the right side of the other, is due to a lesion of the chiasm. If both nasal halves are blind it is called **nasal hemianopsia;** if the two outer halves are blind, it is known as **temporal hemianopsia.** Nasal half-blindness must be due to a lesion of the outer borders of the chiasm, and therefore argues two separate lesions; it is proportionately rare. Temporal half-blindness is due to a lesion involving the inner side of the chiasm, arguing one lesion only, and is more common.

Wernicke's pupillary reaction, i. e., throwing

a beam of light upon the blind half of the retina and observing the pupillary reflex, will determine whether a half-blindness is due to a lesion in front of or behind the geniculate ganglia. If in front, the light reflex of the blind area is absent, because the lesion is then in the path of reflex; if behind, the reflex is present; because at the ganglia the reflex path leaves the optic tract to go to the fourth ventricle.

All varieties of blindness may occur in **hysteria.**

Sudden blindness results from embolism of the central retinal artery, cutting off the entire blood supply from the retina. It occurs in the same conditions which produce apoplexy.

Diminution of vision, as distinct from blindness, is a commoner symptom than blindness in all forms of retinitis, also in degenerations and tumors of the retina, and in anemia, due to poor circulation of the retina.

Scotomata (blind spots) result from some defect in the retina. If the defect is in the peripheral portion of the retina, the spot is not perceived by the patient; if in the central part, the patient sees it as a fixed defect of vision. **Scintillating scotomata,** due to circulatory disturbances, often accompany **migraine.**

Amblyopia, or functional blindness, where no diseases can be demonstrated, and not rem-

ediable with glasses, is due either to the fact that a distinct image has never been thrown upon the retina, or else that the patient has learned to disregard the image. Hence the brain has never learned, or has lost the power, to interpret an image. It is usually associated with errors of refraction and muscle imbalance.

Flashes of light are seen in **retinitis**, due to the sensitive condition of the retina, but steady over-stimulation is almost unknown, because the inflammation quickly renders the rods and cones impermeable to stimulus.

Disturbances of Convergence.

Muscular Imbalance and **Functional Strabismus** are the result of functional inadequacy on the part of one of the extrinsic muscles of the eyeball, associated with errors of refraction, causing either a tendency to deviate, or an actual deviation, of the eyeball in the direction of the opposing muscle or muscles. This condition may be overcome for a time by adaptive innervation of the faulty muscles, but eventually this gives way, and strabismus ensues The inadequate muscle is commonly one of the recti, since these bear the brunt of the work. But it may affect the obliques. Inadequacy of the internal recti causes divergent strabismus; of the external recti, in convergent strabismus.

The imbalance is really due to the fact that the external and internal recti are supplied by separate nerves. The inadequacy of one of them, due to optical defects, is compensated for a time by excessive innervation of both, until the overworked nervous system gives way, and the patient no longer attempts to maintain balance.

Paralytic Strabismus is caused by paralysis of either the third or the sixth cranial nerve, the former causing divergent, the latter convergent strabismus. The lesion may be either in the brain, in the fourth ventricle, or in the course of the nerves themselves.

Functional and Paralytic Strabismus may be readily differentiated by a simple test. In the former, the deficiency of the muscle will manifest itself only when convergence or divergence of the two eyes is attempted; in the latter, it will also manifest itself when conjugate movements of both eyes to right or left is tried.

Luscitas: When the third nerve is paralyzed, not only does it produce divergent squint, but inasmuch as all other muscles which might move the eye are supplied by the same nerve, complete immobility of the eyeball results, known as luscitas.

Conjugate Deviation of the Eye consists in

a deviation of both eyes in the same direction, to right or left, when the patient tries to look straight ahead, and is due to a central lesion which either suspends inhibition in the cranial nerves or paralyzes them. In the former case it is the affected nerve and muscle which becomes spastic, and the deviation is that direction; the eyes "look toward the lesion." In the latter case it is, of course, the unaffected nerve and muscle that get the better so that the deviation is toward the opposite side; the eyes "look away from the lesion."

Conjugate Deviation of the eyes is usually accompanied by a similar deviation of the tongue to a similar involvement of the twelfth nerve.

Disturbances of Accommodation.

Presbyopia: In persons over forty-five years of age, the crystalline lens becomes hard and inelastic, and accommodation is greatly limited. In younger persons and children we sometimes see subnormal accommodation, or premature presbyopia. Its causes are hard to determine.

Paralysis of Accommodation results from (1) central lesions of the fourth ventricle, such as brain tumors, cerebral sclerosis, toxines, etc.; (2) interruptions of the third nerve or the ciliary nerves, as in tumors, neuromia, etc.;

or (3) peripheral paralysis of the ciliary nerves, as exemplified in the use of atropine, and post-acute infectious toxoses. Which of these three causes is the one under consideration must be determined by other symptoms.

THE PUPIL.

The condition of the pupil of the eye, because it is concerned with the delicate reflexes of the iris, furnishes most valuable information regarding the state of the nervous system. It is usually the earliest seat of functional indication of such disease.

Sluggish or immobile pupil results either from (1) adhesion of the iris to the lens, as in iritis with exudation, or (2) disease of the central nervous system, e. g., cerebral syphilis, tumor of the brain.

Unequal pupils are practically always pathognomonic of lesions on one side or other of the brain.

Mydriasis (dilation of the pupil) is caused by any condition which (a) paralyzes either the central or peripheral end of the third nerve, (b) inhibits its functionation through the brain, or (c) stimulates the sympathetic.

Tumors of the brain, and of the third nerve, and the action of certain drugs, notably atropin, are examples of the first class.

Fevers, comatose conditions, hemasthenia, tuberculosis, and nervous depression are instances of the second class.

Pain, sensory irritation, and visceral diseases furnish illustrations of the third variety.

Myosis (contraction of the pupils) on the other hand, results from influences which (a) irritate the third nerve, or (b) depress the sympathetic.

Photophobia, from any cause, brain tumors, meningitis and certain drugs, as eserin, are instances of the former; spinal sclerosis, aneurisms (by pressure) and opium, of the latter.

Argyll Robertson Pupil, in which the pupil reflex is wanting, is seen in tabes dorsalis and dementia paralytica, supposed to be due to involvement of Minert's fibres.

Wernicke Pupillary Reaction is a test for determining the location of an injury to the optic tract (usually employed in cases of hemianopsia). If the reflex is present, in the half of the eye that is affected, then the lesion is back of the corpora quadrigemina; if not, it is in front of these ganglia; because at that point the reflex path leaves the optic tract to go to the ventricle.

Hippus, a periodic contraction and dilatation of the pupil, due to alternate excitation

and inhibition, is seen in disorders affecting the emotions, as in mania, hysteria, etc.

Inter-Ocular Tension results from a disturbance between the secretion and absorption of the humors. This condition is known as **Glaucoma.**

The steady increase of internal pressure in this disease produces a characteristic train of symptoms, such as steady pain, cupping of the disc, vascular stasis, dimness of vision, and, if not relieved, eventual blindness due to destruction of the eye.

Scotoma (blind spot) is frequently due to opacities in the humor, especially the vitreous, in which case they are seen by the patient as spots before the eyes (muscae volitantes).

Cataract, an opacity of the lens, produces, of course, diminished acuteness of vision, most marked when it is central, least marked when it is peripheral in location. Vision is better in dim light, because the pupil is then dilated and more rays (relatively) enter the eye.

Increase in index of refraction, due to increased density of the lens, occurs in cataract producing **myopia** (short sight).

Color Blindness: Frequently a patient is met with who is unable to perceive certain colors, and is therefore said to be color blind

to that particular color and its combinations. The commonest colors to which color blindness is found are red and green. This condition may be (a) retinal, i. e., due to a defect of the retina, by far the most common variety, or (b) central, i. e., due to a defect in the visual center. Whichever of the two theories above outlined be accepted, the retinal defect must consist in an absence of the photo-chemical substance whose maximum modification is brought about by the spectrum wave corresponding to the color to which sensation is lacking.

Because of its investigation by Dalton, this condition is sometimes known as Daltonism.

SPEECH.

Disturbances of Speech: Aside from aphasia, which has been discussed elsewhere, the act of speech is subject to two general types of disturbance from impairment of the neuronic tract, (a) Precipitate Speech, (2) Scanning Speech.

Precipitate Speech, in which the words tumble over each other in their haste to be spoken, is a phase of incordination due to suspension of the inhibiting faculty of the brain, with or without loss of intelligence. It is seen in organic diseases of the brain, such as dementia

paralytica, general paresis, thrombosis, in their early stages, while as yet the actual motor mechanism of speech is unimpaired; and in psychosis, such as hysteria, mania, etc.

Scanning Speech, in which every syllable is distinctly uttered, with an appreciable interval between them, is a phase of intention tremor, due to partial impairment of the motor tract of speech, with or without loss of intelligence. It represents the attempt of the brain, conscious of the defect in its motor mechanism, to avoid incoherency, and is akin to the slow, careful, pseudo-spastic gait of conscious ataxia. It is seen in diseases that involve the motor neurons concerned in speech, of which disseminated sclerosis is the most conspicuous example.

HEARING.

Increased Hearing: In nerve lesions which involve the seventh nerve, whether centrally or along its course, provided it be above the point in the Fallopian canal where the stapedius nerve goes to the ear, the hearing is increased in acuteness, because when this branch is paralyzed the chorda tympani goes into permanent dominance, and the drum is constantly tense.

Decreased Hearing (deafness) on the other hand, results from any lesion of the fifth nerve,

centrally or above the branching of the tensor tympani, because of precisely the opposite conditions to those described above. Deafness also results, of course, from any lesion of the auditory nerve itself, either at its centre in the temporal lobe, or along its course, such as tumors, hemorrhages, scleroses, inflammations, etc.

Much more commonly deafness is due to imperfect vibration of the tympanic membrane, caused by conditions which (1) interfere with the diaphragmatic adjustment of the membrane, as in myringitis (inflammation of the drum), chalky deposits on the drum, catarrh, impaction of the cerumen, all of which thicken the membrane; perforation, which destroys its tension and integrity; and blocking or stenosis of the Eustachian tube, which interferes with air pressure and causes the drum to sag or bulge, as the case may be; and (2) block the meatus leading to the drum, as in catarrh, impacted wax, and growths of the meatus.

Deafness, again, results from anything which either changes the relative sizes and relations of the ossicles of the middle ear, such as hypertrophy due to catarrh, caries, etc., or which interferes with their free motion, as ankylosis, otosclerosis, etc.

Rinne's test for determining whether the

cause of deafness is located on the outer or inner side of the fenestra ovalis, i. e., whether it is in the middle ear or outer ear, on the one hand, or in the inner ear or brain on the other hand, is to vibrate a tuning fork and hold it close to the opening of the meatus, then vibrate it again and place the stem on the tip of the mastoid, and with a second-watch observe by which method the patient hears the sound the longest. If the course from the external meatus to the fenestra is interrupted, while the internal ear and auditory nerve are intact, vibrations will be communicated to the perilymph better, and heard longer, by way of the bone than by the meatus (Rinne+), whereas if the opposite be the pathological condition, the sound is better conducted and longer heard by way of the meatus (Rinne —).

Weber's Test is to vibrate a tuning fork and hold the stem upon the vertex or crown of the head. If the lesion is in the middle or outer ear, the sound heard louder in the deaf ear, because, with the middle or outer ear blocked, the vibrations received through the head bones find no outlet through the meatus, and are reflected back into the perilymph, thus intensifying the sound (Weber +).

In **diseases of the labyrinth** extending from the middle ear, the hearing for high pitched

notes is greatly impaired, because the few short rods at the commencement of Corti's organ, hear the vestibule resonate to high pitched sound waves.

Auditory Limits: The range of perceptible sound waves varies in different individuals, but the average audible gamut is from 30 vibrations per second (low pitch) to 40,000 per second (high pitch). Slower vibrations than the former, if perceived at all, are usually only perceived as stimulations of the sensory nerve of the tympanum—the auditory nerve does not react to them; or else the auditory nerve responds to their overtones, for particulars of which a work on acoustics must be consulted.

Pathological Limitations of Pitch are caused by ankylosis of the ossicles which prevents those bones from vibrating rapidly.

The auditory nerve collects the vibrations from the rods of Corti and transmits them to the auditory centre in the temporal lobe, each ear being separately represented in each corresponding temporal lobe.

Sudden Deafness is a prominent symptom of **Meniere's Disease,** caused by a hemorrhage into the auditory nerve or labyrinth.

Tinnitus is a symptom of all ear disorders which increase tension in or shut off egress

from the internal ear, because the physiological noises, such as circulation, muscle tonus, etc., which under ordinary conditions are too diffused to be heard, are confined to the labyrinth and become audible.

Vertigo and Dizziness accompany all such conditions because of the interference with the integrity of the semi-circular canals and consequent disturbances of coordination. (See Nervous System.)

Auditory Centre: The auditory centres in the temporal lobes transmit the sensation of sound to the auditory centre in the left frontal.

Affections of the auditory centre are fully dealt with under cerebrum.

SMELL.

Anosmia (loss of smell) is, of course, seen in all disorders which (a) impair or destroy the mucous membrane in which the filaments are spread, as in rhinitis, especially the chronic hypertrophic and atrophic forms, catarrh, etc., or (b) obstruct the nares, as in adenoids, polypi, hypertrophy or the turbinates, new growths and allied troubles.

Anosmia results from any condition which (a) destroys or impairs the path of innervation, or (b) renders the nerve or centre irresponsive to stimuli. Brain tumors and cere-

bral softening are the most conspicuous examples of the first; hysteria, melancholia and nervous depression of the second.

Hyperosmia (increased sensitiveness to smell) and parosmia (perverted sense of smell) are usually nervous disorders (vide infra).

Hyperosmia is usually an accompaniment of hysteria, which renders the olfactory centre unusually acute.

Parosmia is also the result of such functional psychic aberrations as hysteria, neurasthenia, and melancholia, but may be due to congenital absence of certain specific fibres in the nerve.

TASTE.

Absence or Dinimution of Taste results from any condition which destroys or impairs the mucous membrane in which the nerve filaments or taste buds lie, as cancer, ulceration, tuberculosis, and severe forms of glossitis.

N. B.—Absence of taste, so-called, in catarrh, is due to impairment of retronasal olfaction.

Absence of Taste also results from any condition which interrupts the course of the nerve currents concerned, or the recording of those impulses in the brain, as brain tumors, neuromata, neuritis, etc.

Hypersensitive Taste is seen in conditions which exalt the nervous functions, as hysteria, and the influence of certain drugs, strychnia, etc.

Perversion of Taste (far more frequent than diminution) may arise from conditions of the tongue and palate which alter the sapid substance, as salivary disorders (mumps) digestive troubles (from food coating), fevers and sore throat (from epithelial coating), etc. The anomaly is, therefore, not really a perversion of the sense, but of the sapid substance.

True perversion of taste is almost always due to perversion of psychic function, as in melancholia, hysteria, neurasthenia, hypochondria, etc.

Dissociated Taste Sensations are diagnostic of disorders of the tongue occurring in patches, which can be located (but not with exactness) by the dominant sensations.

REPRODUCTION.

Ovulation.

Azoospermatism, an absence of spermatozoa in the semen, results from secretory disablement of the testicles. Absolute azoosperma is a rare condition, requiring complete disablement of both testes, but comparative azoosperma is not infrequent, consisting of a dimi-

nution in number or activity of spermatozoa. It is seen in malignancy of the testicle, orchitis, syphilis, tuberculosis, and sexual exhaustion from excessive coitus. Its effect is, of course, total or relative sterility.

Azoospermatism is sometimes seen in conditions which impair the patency of the vasa deferentia, preventing the passage of the spermatozoa into the vesicles.

In **seminal vesiculitis,** especially of the chronic type, the walls of the vesicles frequently become adherent, and the spermatozoa are unable to pass out into the semen. Such a condition, of course, produces asperma and sterility.

Prostatitis, especially when chronic, often results in sterility, due to the absence of prostate fluid, and consequent immotility of the spermatozoa. This condition of the semen is known as colloid semen.

Absolute sterility in the female, such as occurs in the male in the absence of the testicular secretion, occurs in the female in total disability of both ovaries. Fortunately such a condition is exceedingly rare, and is only found in such uncommon cases as bilateral ovarian cysts, ovarian atrophy and profound constitutional diseases in which the ovarian vascularity suffers in common with the other organs. As

in the case of the male, however, sterility from complete suppression of ovulation is extremely rare.

Sterility is more frequently due to disease of the tubes than to all other causes combined. Any condition which impairs the integrity of the tubes naturally hinders the passage of the ovum and prevents its meeting with the male element. Conspicuous examples are seen in salpingitis, hydro- and pyo-salpinx, and tubercular infiltrations of the tubes.

Excessive ovulation results from a hyperemic state of the ovary, such as is induced by frequent coitus, ovaritis, and any other form of pelvic congestion. In such cases the vitality of the rapidly matured ovum is below par, so that the net result of such conditions is diminished fertility, or relative sterility.

Premature Menopause is induced by those conditions which unduly increase ovulation, owing to the rapid exhaustion of ovarian vitality.

Ectopic Gestation of the abdominal type occasionally occurs as the result of the failure of the ovum to reach the tube, and the migration of a male sperm-cell into the abdominal cavity, where the two meet and fuse.

Amenorrhea is commonly understood to signify simple failure of the visible flow in the

second period of menstruation, and as such may result from obstructive and anatomic causes. True functional amenorrhea, however, is a failure of the whole process, and usually depends upon (a) some constitutional dyscrasia by which vascularity of the generative organs suffers with that of the other organs, or (b) some local vaso-motor disturbance in the generative tract.

Of the former variety, anemia, tuberculosis, and neurasthenia are frequent examples; of the latter type, trauma, surgical shock, cold, etc.

Menorrhagia (excessive flow), on the other hand, results from those conditions which increase uterine congestion and metabolism, either systemically, as in fevers, or locally, as in metritis, local malignancy, and all forms of pelvic inflammation.

Dysmenorrhea, outside of those cases due to anatomic anomalies, is comparatively rare. True functional dysmenorrhea is usually due to a neurosis of some kind, as neuralgia, neurasthenia, or hysteria.

Suspended Menstruation with no other signs of disturbed health, especially in a woman who has heretofore been regular, is almost infallibly indicative of pregnancy.

Vicarious Menstruation: Sometimes the mucous membrane of the uterus fails to under-

go degeneration, and under the increased vascular tension the capillaries in other parts of the body, such as the breast, stomach, lungs, nose, etc., break down in hemorrhage.

Supplementary Menstruation occurs when the capillaries of other organs break down in addition to uterine disintegration.

FERTILIZATION.

Impotence, as to **erection,** results from any condition which (a) interrupts the course of the reflex, or (b) inhibits it from the higher centres.

The former conditions are found in all of those spinal diseases which impair the integrity of the lumbar centre, as tabes dorsalis, sclerosis myelitis, and in all of those nervous diseases in which the general reaction to stimulus is lowered, as neurasthenia, tuberculosis, diabetes, etc.

The latter type of impotence is generally known as psychical impotence, and is seen in hysteria, melancholia, and neuroses of all kinds.

Impotence, as to **ejaculation,** depends in a general way, upon essentially the same functional disturbances as failure of erection, and the classification under that head may be accepted as applying to this process. It may be said, however, that this faculty fails earlier

than that of erection; and to other causes must be added, of course, those conditions in which semen is secreted. The latter, however, do not of themselves really influence the functional performance of ejaculation, as the reflex takes place even though no fluid is ejaculated.

Priapism, on the other hand, arises from any state which (a) renders the reflex abnormally sensitive, or (b) stimulates the cerebral end of the tract.

In the former class are inflammatory spinal diseases, as early myelitis, meningitis, spinal and cerebral hyperemia and growths. In the latter class are manias, epilepsias, hysteria, etc.

Ejaculation is accomplished by a vigorous and sudden contraction of the muscles of the vaso deferentia, seminal vesicles, perineum and urethra, in the sequence indicated, throwing the semen into the female vagina. It is the last part played by the male in the process of reproduction.

Innervation of Ejaculation is similar to that of erection, is effected through the same spinal centre, and stimulated by an intensification of the same peripheral stimuli. It is, however, rarely precipitated by direct cerebral stimulation, as erection is.

Premature Ejaculation and Emissions occur

under the same conditions as those enumerated under Priapism, q. v.

Infecundity, due to impaired integrity of the tubes, is, as already stated, far more frequent than from any other cause. Any condition which renders the tube impassable and prevents the passage of the male spermatozoon is fatal, of course, to impregnation. Such conditions are found in plastic adhesions caused by salpingitis (especially gonorrheal), tubal tuberculosis, and in pyo-, hydro-, and hematosalpinx.

Tubal Pregnancy: In some instances the impairment of the tubes, although not sufficient to hinder the passage of the spermatozoon, is enough to prevent the fertilized ovum from passing down into the uterus, in which case it remains in the tube and there develops into a fetus. This is known as **tubal ectopic pregnancy.**

GESTATION.

Incapacity for Gestation (barrenness) may arise from any condition of the uterus which renders it unfit for the attachment of the placenta. Such incapacity usually results from either an anemic and poorly nourished state of the uterus, or in constitutional diseases, malignancy, anemia, or from opposite condition

of hyperemia, as in metritis, endometritis, pelvic congestion, tumors, etc.

Abortion due to the premature severing of the ovum from the uterine wall often results from the same causes as those just enumerated and for the same reason.

Vomiting of Pregnancy: In the early weeks of gestation the volume of maternal blood has not yet adjusted itself to the increased demands upon it. The consequence is an impoverished and toxic condition, of the maternal blood which poisons and irritates the nerve centers and causes nausea and vomiting. "Morning sickness" is the expression of a cerebral anemia due to assuming the vertical posture after sleep.

Death of the fetus, of course, follows interruption of the communication between the maternal and the fetal organism—in other words, any impairment of the integrity of the placenta.

Abortion, in the sense of premature termination of gestation, occurs more frequently from placental impairment than from any other cause outside of mechanical trauma.

Placental Impairment: As the placenta is essentially a vascular organ it is subject to all of the disorders that affect other vascular organs. Such disorders may be (a) of a con-

stitutional nature, affecting the placenta in common with the rest of the organism, or (b) local in character, peculiar to the placenta. Of the former, syphilis is by far the most common offender; tuberculosis, malignancy, diabetes, nephritis, toxemia (of any kind), all of which degenerate the placental tissues; in the latter variety are included embolism, thrombosis, and apoplexy of the placenta, hydatid pregnancy, infarcation, calcareous and fatty degenerations, etc.

Infection may pass in either direction, from mother to fetus, or from fetus to mother, through the placental medium. In this way syphilis, tuberculosis and other infectious diseases are frequently transmitted.

Malnutrition: Those placental conditions cited as causes of fetal death commonly are not sufficient to kill the fetus, but result in its insufficient or vitiated nutrition.

PARTURITION.

Protracted and Precipitated Labor are the two opposite deflections from the normal process, depending upon a disturbance of the normal balance between the uterine forces and the dimensions of the fetus.

Protracted Labor is due either to a subnormal character of uterine contractions, or to

a super-normal size of the fetus. The former is known as uterine inertia, and is seen in all conditions of poor constitutional health, and in myoma of the uterine walls, due to tumors, fatty degeneration, anemia, tuberculosis, etc.

Precipitate Labor results from either abnormally powerful uterine contractions or an abnormally small fetus, usually the latter.

Obstructive and anatomic causes of dystocia are not functional in their nature and will not be discussed here.

Premature Parturition (abortion) results, outside of trauma and physical accidents, from any condition which sets in motion the stimulus above referred to before the fetus is matured. Until more is definitely known of the nature of the stimulus, little can be predicated about its premature excitation.

Accepting the theory that the normally matured fetus acts as an independent foreign body in the uterus, then any condition which renders the fetus an independent foreign body—in other words, anything which terminates the physiological union of mother and fetus, causes expulsion. Among such conditions are constitutional disease, causing placental degeneration, hyperemia of the pelvis from exercise or coitus, placental apoplexy.

Mental shock may also start premature contractions through the cerebro-spinal tract.

LACTATION.

Agalactia, absence of milk, in its complete form, is extremely rare, but relative agalactia is not uncommon, depending, of course, on either a disability of the glands themselves, as in malignancy, anemia (lack of vascular nourishment), diabetes, tuberculosis, syphilis (lack of cellular nutriment), and mastitis; or to the impairment of the nervous mechanism, as in anomalies of the uterus and ovaries (perverted stimulus), and more frequently in mental disturbances such as melancholia, neurasthenia, hysteria, shock.

Polylactia, an excess of milk, is very common, and results from any condition which (a) increases the flow of blood to the breasts, or (b) overstimulates the nervous mechanism. In the former class are mastitis, vaso-motor disturbances, etc. In the latter, metritis, ovaritis and psychic influences.

Premature Lactation is precipitated by a premature consummation of physiological conditions which normally mature at parturition. It usually indicates premature separation of the fetus from physiological dependence upon the mother, hence fetal death.

The milk has an average composition as fol-

lows: Proteids, 1 to 2 per cent.; fats, 3 to 4 per cent.; sugar, 6 to 7 per cent.; salts, .01 to .02 per cent. There are also negligible quantities of nitrogen, urea, creatin, lecithin, cholestrin, etc.

The bulk of the proteins are in the form of casein. This and the milk sugar are true secretory products of the mammary glands, as neither pre-exists in the maternal blood.

Abnormal Proportions of the constituents of the milk occurs in cases where the maternal metabolism exhibits corresponding inequalities. Thus when protein metabolism predominates the proteins in the milk are high, and vice versa.

Abnormal proportions of the constituents of the milk are seen in cases where the maternal metabolic balance exhibits corresponding unequalities. Thus, when the nitrogen balance is plus the proteins in the milk are high, and vice versa.

Abnormal Composition: As the excreting cells of the breast are easily permeable to any constituent of the blood that is in excess of normal, the milk may and frequently does contain toxic products existing in the blood as a result of disease.

Mental Influences powerfully affect the constituency of the milk by perverting the innervation of the process.

ENCEPHALITIS, ACUTE.

Etiology.	*Pathology.*	*Symptoms.*	*Treatment.*
Micro-organic infection. Traumatism. Attacks gray matter of brain. Rare, because of protected location and anastomosis of brain.	Bacterial invasion. Absorption of toxins.	Rapid onset. Chill. Fever (103-104°). Rapid pulse.	Serum and vaccine, if nature of infection is known.
	Irritation of cortex by congested vessels and minute hemorrhages.	Headache. Twitchings. Curled-up posture. Irritability. Delirium. Projectile vomiting.	Derivative and revulsive: Heat to extremities. Purge with Elaterine, Veratrum, Aconite, Atropine. Antispasmodic: Gelsemium, Hyoscine.
	Later: Exudation of serum into cerebral interspaces, causing compression.	High temperature (106-107°). Slow pulse (40-50). Pin-point pupils. Coma. Diminished reflexes.	Absorptive and revulsive: Lobelia, Apocynum, Blue Mass. Purge with Elaterine.

ENCEPHALITIS, SUPPURATIVE (BRAIN ABSCESS).

Etiology.	*Pathology.*	*Symptoms.*	*Treatment.*
Mixed infection. Primary or metastatic tuberculosis with pyogenic grafting.	Absorption of toxins.	Temperature 100-101°. Rapid pulse. Leucocytosis.	Vaccine if nature of infection is known. Nuclein to aid leucocytosis.
	Intracranial pressure and irritation.	Persistent localized headache. Unequal pupils. Projectile vomiting. Reflexes exaggerated. Percussion. Dullness. Muscles spastic. Optic neuritis.	Surgical drainage, if available.
	May be encapsulated or diffused.	Focal symptoms according to site of lesion.	
	May rupture into brain, or gradually infiltrate gray matter.	Death from apoplexy or coma.	

MENINGITIS, ACUTE (SIMPLE INFECTIVE).

Etiology.	*Pathology.*	*Symptoms.*	*Treatment.*
Micro-organic infection. Traumatism. Exciting causes: Exanthemata, syphilis.	Micro-organic invasion. Absorption of toxins.	Chill. Fever (102-103°). Rapid pulse.	Serum and vaccine if nature of infection is known. Nuclein to aid leucocytosis.
	Irritation of healthy brain and cord by congested meninges.	Violent headache (characteristic). Muscular twitchings. Opisthotonos. Delirium. Projectile vomiting. Strabismus and ptosis. Spastic reflexes. Babinski reflex. Dilated pupils.	Derivative and revulsive. Cold to head, heat to extremities. Purge with Elaterine. Veratrum, Aconite, Atropine. Antispasmodic: Gelsemium, Hyoscine.
	Later: Profuse outpouring of serous fluid on brain, causing great compression.	Hyperpyrexia. Slow bounding pulse. Shallow labored breathing. Profound coma. Pin-point pupils. Diminished reflexes.	Revulsive: Lobelia, Apocynum, Blue Mass. Guard the heart with stimulants. Spinal puncture.
	Usually spreads to and violates brain cells.	Usually leaves some cerebral impairment.	Reconstructive: Phosphorus, Fowler's Solution.

Etiology.	*Pathology.*	*Symptoms.*	*Treatment.*
Virulent micro-organism.	Bacterial invasion. Absorption of toxins.	Violent onset. High fever (104-105°). Rapid, full pulse. Vomiting. Leucocytosis (high).	
Attacks meninges of brain and cord.			
	Hemorrhagic irritation of brain and cord by intensely inflamed meninges.	Violent pain in head and neck. Opisthotonos. Convulsion or trismus. Spastic reflexes. Babinski test. Hyperesthesia of senses. Lumbar puncture.	Derivative and revulsive: Cold to head, heat to extremities. Purge with Elaterine. Veratrum, Aconite, Atropine. Antispasmodic: Gelsemium, Hyoscine. Withdraw 20 to 30 c.c. of cerebro-spinal fluid and inject same quantity of Flexner's serum. Nuclein to aid leucocytosis.
	Within 48 *hours*: Profuse outpouring of bloody serous fluid on brain and cord, causing great compression.	Hyperpyrexia. Slow bounding pulse. Profound coma. Pin-point pupils. Diminished reflexes. Death usually occurs in three to five days in spite of treatment.	Revulsive: Lobelia, Apocynum, Blue Mass. Guard the heart with stimulants. Spinal puncture.

MENINGITIS, TUBERCULAR (HYDROCEPHALUS)

Etiology.	*Pathology.*	*Symptoms.*	*Treatment.*
Tuberculosis. Attacks meninges of brain chiefly.	Absorption of tubercular toxins by meningeal vessels.	Slow onset. Slight fever (101-102°). Rapid, soft pulse. Diarrhea. Slight leucocyte increase.	Nuclein to increase leucocytosis.
	Diffusion of tubercles through meninges, and poisoning of nerve tissue.	Dull headache. Stupor. Muttering delirium. Spasms. Hydrocephalic cry. Projectile vomiting. Spastic reflexes. Retraction of muscles of neck and abdomen. Babinski test.	The treatment of the underlying tuberculosis. Vigorous derivative treatment is contraindicated.
	Later: Slow infiltration of brain and cord with serous exudates.	Gradual deepening of coma. Pin-point pupils. Diminished reflexes. Choked disc and tubercles. Death occurs in spite of treatment.	Absorptive: Inunctions of Blue Mass and Iodides.

MYELITIS, ACUTE.

Etiology.	*Pathology.*	*Symptoms.*	*Treatment.*
Micro-organic infection. Traumatism. Exposure. Constitutional disease (syphilis).	Invasion of germs. Absorption of toxins.	Sudden onset. Chill. Fever (102-103°). Rapid pulse.	Vaccine and serum if nature of infection is known. Nuclein to increase leucocytosis.
	Irritation of spinal foci by minute hemorrhages and toxines.	Lightning pains. Jactitations.	Derivative and Revulsive: Cold to head, heat to extremities. Purge with Elaterine. Veratrum, Aconite, Atropine. Antispasmodic: Gelsemium, Hyoscine.
	Infiltration of spinal gray matter.	Bilateral anesthesia below lesion. Bilateral paralysis below lesion.	
Cervical, dorsal or lumbar, according to segments involved.	Diseased process demarked from healthy portion.	Girdle sensation.	

MYELITIS, ACUTE (Continued).

Etiology.	*Pathology.*	*Symptoms.*	*Treatment.*
	Sensory paths impaired.	Ataxia.	
	Meinert's fibres involved.	Argyle-Robertson pupil (in cervical myelitis).	
Diffuse Form: Attacks entire section of cord.	Roots of lower neurones involved.	Reflexes diminished. Muscles flaccid. Atrophy rapid. R. of D. present. Dragging gait.	Galvanism to affected muscles. Reconstructive: Phosphorus and Neuro-Lecithin.
Transverse Form: Attacks only transverse gray matter of cord.	Only transverse (upper) neurones involved.	Reflexes exaggerated. Muscles spastic. Atrophy slow. R. of D. absent. Spastic gait.	Galvanism to affected muscles. Reconstructive: Phosphorus and Arsenic.

ACUTE POLIOMYELITIS (INFANTILE PARALYSIS).

Etiology.	*Pathology.*	*Symptoms.*	*Treatment.*
Specific micro-organisms (not yet isolated).	Bacterial invasion. Absorption of toxins. Violent hyperemia of gray matter of brain and cord. Violation of Neurons.	Chill. Fever (103-104°). Rapid pulse. Violent pains. Muscular twitchings followed by rapid paralysis.	Derivative and revulsive: Cold to head, heat to extremities. Purge with Elaterine. Veratrum, Aconite, Atropine. Antispasmodic: Gelsemium, Hyoscine. Biologic: Nuclein to raise resistance.
	Encephalitic Form: Attacks brain.	Headache. Twitchings. Curled-up posture. Irritability. Delirium. Projectile vomiting.	Galvanism to affected muscles. Reconstructive: Phosphorus and Fowler's Solution.
	Meningitic Form: Attacks meninges.	Violent headache (characteristic). Muscular twitchings. Opisthotonos. Delirium. Projectile vomiting. Strabismus and ptosis. Spastic reflexes. Babinski reflex. Dilated pupils.	

ACUTE POLIOMYELITIS (INFANTILE PARALYSIS)—Continued.

Etiology.	*Pathology.*	*Symptoms.*	*Treatment.*
	Spinal Form: Attacks grey matter of cord, especially anterior horns.	Lightning pains. Jactitations. Bilateral anesthesia below lesion. Bilateral paralysis below lesion.	
	Usually a mixture of the three forms, with one predominating.	Atypical, asymmetrical, irregular.	
	Always leaves some permanent injury to neurons.	Residual paralyses. Contractures. Atrophies. Spasticities. Flaccidities.	Braces, tenotomies, transplantations, etc., by orthopedic surgeon.

ACUTE ASCENDING PARALYSIS (LANDRY'S DISEASE).

Etiology.	*Pathology.*	*Symptoms.*	*Treatment.*
Micro-organic infection. Traumatism. Exposure.	Micro-organic invasion and absorption of toxins.	Very rapid onset. Chill. Fever (101-102°). Rapid pulse.	
It is a special form of acute myelitis.	Irritation of foci by toxins and minute hemorrhages in spinal roots.	Lightning pains (beginning in legs). Jactitations.	Same as in myelitis, but unavailing.
	Extension of process by ascending degeneration, rapid and complete.	Rapid and complete paralysis, beginning in legs and rapidly involving trunk and arms. Absent reflexes. Flaccid muscles (very).	
	Involvement of bulbar neurons.	Death by paralysis of respiration. Rapid course.	

MULTIPLE NEURITIS, SIMPLE INFECTIVE.

Etiology.	*Pathology.*	*Symptoms.*	*Treatment.*
Infection by micro-organisms.	Invasion and multiplication of germs and toxins.	Malaise. Chill. Temperature (102-103°). Pulse 120-130.	Nuclein to raise resistance.
Attacks lower neurons. *i.* e., spinal nerves of trunk and limbs.	Irritation of nerve tissue by inflamed vessels and minute hemorrhages.	Pain (constant and severe). Jactitation.	Derivative: Rest Heat. Veratrum, Aconite, Atropine.
	Exudation into nerve tissues.	Anesthesia. Paralysis.	Absorptive: Heat, Ergot to tone up vessels. Reconstructive: Arsenates, Massage, Galvanism.
	Involvement of lower neurones, cutting off spinal current.	Reflexes diminished. Muscles flaccid. Atrophy rapid. R. of D. present.	Faradism and Galvanism to affected muscles.
	Impairment of sensory paths.	Ataxia.	
	Involvement of bulbar neurones.	Suffocation. Dysphagia (rare).	

Etiology.	*Pathology.*	*Symptoms.*	*Treatment.*
Alcoholism.	Alcoholic toxemia and absorption.	Gastritis. Vomiting. Rapid weak pulse.	Treatment of acute alcoholism.
Usually attacks all lower neurones of limbs and trunk.	Irritation of nerve tissue by congested vessels and small hemorrhages.	Pain (constant and severe). Jactitations.	Derivative: Heat, rest. Veratrum, Aconite, Atropine.
	Exudation into nerve tissue.	Anesthesia. Paralysis.	Absorptive: Heat, Ergot to tone up vessels. Reconstructive: Arsenates, Massage, Galvanism.
	Lower neurones involved, but modified by lack of brain inhibition due to alcoholism.	Reflexes diminished in strength, but spastic in quality. Muscles flaccid but often masked by edema. Atrophy rapid. R. of D. present.	Faradism and Galvanism to affected muscles.
	Both brain poisoning and impairment of sensory paths.	Ataxia profound.	
	Extension to bulbar neurones.	Frequently paralysis of respiration.	

CEREBRAL CONCUSSION.

Etiology.	*Pathology.*	*Symptoms.*	*Treatment.*
Hard blow on skull.	Momentary compression of brain (pneumatic).	Unconsciousness. Dilated pupils. Temperature subnormal. Pulse, usually weak and slow.	Mild stimulation. Heat to extremities.
	Return to normal.	Returning consciousness. Vomiting of blood (swallowed during unconsciousness). Pulse bounding. Slight rise of temperature.	Expectant treatment to avoid inflammation. Atropine, Veratrum, Digitalis, but do not over-do. Quiet, and stop diet.

CEREBRAL ANEMIA.

Etiology.	*Pathology.*	*Symptoms.*	*Treatment.*
General anemia. Malaria. Aortic stenosis. Arteriosclerosis. Shock. Hemorrhage. Ligature of carotid.	Brain is deficient in blood supply.	Pallor. Vertigo. Syncope. Dimness of vision. Dilated pupils. Pale disc. Frequent yawning. Intense headache (often one-sided). Irritability.	Treat underlying cause. Reconstructive: Fowler's Solution. Nuclein. Generous Diet.

CEREBRAL CONGESTION, PASSIVE.

Etiology.	*Pathology.*	*Symptoms.*	*Treatment.*
Exposure to sun. Brain work. Gastric irritation. Alcohol. Cardiac disease. Venous obstruction. Suppression of menses.	Stasis of blood in cerebral vessels.	Dull headache (general). Vertigo. Photophobia. Bloodshot eyes. Tinnitus aurium. All symptoms increased by recumbent posture.	Remove cause. Derivative: Atropine. Heat to extremities. Sharp catharsis. Light diet. Exercise.

GENERAL PARESIS.

Etiology.	*Pathology.*	*Symptoms.*	*Treatment.*
Syphilis, alcohol. Attacks psychic and motor areas of brain.	Slight changes in pia and molecular layer of brain.	Failing memory for essential things. Moral sense blunted. Hesitation of speech. Fine tremors. Wassermann test positive with spinal fluid. Spirochetae in spinal fluid.	No treatment availing. Keep gastro-intestinal tract antiseptic.
	Destruction of upper brain layers by sclerosis.	Amnesia. Agraphia. Incoherence. Spasms.	Phosphorus to nourish nerve-tissues.
	Involvement of motor cortex (upper neurones).	Reflexes exaggerated. Babinski test positive. Muscles spastic. Tremors.	Galvanism and Hyoscyamine as antispasmodics.

BRAIN TUMOR.

Etiology.	*Pathology.*	*Symptoms.*	*Treatment.*
In order of frequency: Tuberculous, Gliomatous, Sarcomatous, Cystic, Gummatous.	Intracranial tension from hard, fixed tumor.	Slow, full pulse. Headache, severe and persistent. Unequal pupils. Projectile vomiting. Exaggerated reflexes. Choked disc. Dull area of skull percussion.	Surgical removal, if possible.
	Fixed location.	Focal symptoms according to site of lesion. Very marked.	
	May enlarge or cause general degeneration of gray matter.	Death from coma or exhaustion.	

BRAIN HEMORRHAGE (APOPLEXY).

Etiology.	*Pathology.*	*Symptoms.*	*Treatment.*
Arteriosclerosis. High vascular tension.	Rupture of artery into brain substance.	Sudden loss of consciousness and motor power of varying extent.	Mildly revulsive: Atropine in small doses; Aconite, Veratrum and Digitalis. Cold to head and neck. Heat to extremities.
	Extravasated blood partially absorbed.	Usually return to consciousness and partial regain of motion and sensation.	Mild stimulation: Tea and ammonia.
	Organized clot on brain substance, with partial degeneration.	Partial paralysis according to site of lesion. Exaggerated reflexes. Optic neuritis. Unequal pupils. Weakened mentality. Conjugate deviation of eyes. Stertorous breathing. Pulse slow and bounding.	Absorptive and reconstructive: Mercury Biniod., Phytolacca, Iodoform and Arsen. Iod. in small doses. Colchicine to unload effete matter. Fowler's Solution and Nuclein to reconstruct blood.

CEREBRAL EMBOLISM.

Etiology.	*Pathology.*	*Symptoms.*	*Treatment.*
Ulcerative endocarditis. Fatty degeneration. Valvular disease. Usually attacks terminal artery of brain.	Sudden plugging of cerebral artery, with shock and anemia of areas involved.	Sudden onset. Complete or partial unconsciousness and hemiplegia. Unequal pupils. Aphasia (frequent).	Mild stimulation.
	Absorption of embolus or collateral circulation.	Marked improvement of symptoms.	Absorptive and reconstructive: Mercury Biniod., Phytolacca, Iodoform and Arsen. Iod. in small doses.
	Certain proportion of brain tissue irreparably damaged.	Recovery never complete.	Colchicine to unload effete matter. Fowler's Solution and Nuclein to reconstruct blood.

CEREBRAL THROMBOSIS (SOFTENING)

Etiology.	*Pathology.*	*Symptoms.*	*Treatment.*
Arteriosclerosis. Endocarditis.	Gradual diminution of cerebral arterial lumen, with resulting anemia of brain areas involved.	Gradual onset. Vertigo. Monoplegias. Paresthesia. Torpid pupils. Aphasia (partial). Mental dullness. Soft weak pulse. Vomiting. Exaggerated reflexes. Spastic muscles.	Absorptive and reconstructive: Mercury Biniod., Phytolacca, Iodoform and Arsen. Iod. in small doses. Colchicine to unload effete matter. Fowler's Solution and Nuclein to reconstruct blood.
	Anemic areas break down and degenerate, softening or sclerosing.	Progressive deepening of symptoms. Extension of paralysis. Coma. Death.	

MULTIPLE SCLEROSIS.

Etiology.	*Pathology.*	*Symptoms.*	*Treatment.*
Micro-organic infection. Traumatism. Attacks neurones of brain and cord, especially cervical segments.	Gradual sclerotic degeneration scattered in disseminated foci through gray matter of brain and cord, disabling (but not completely) the neurones and irritating them.	Onset gradual. Dull headache. Twitchings. Intention tremors. Nystagmus. Hippus. Slow scanning speech.	Alterative and antispasmodic: Chromium sulphate to check hyperplasia. Hyoscyamus and Gelsemium as antispasmodics. Nuclein to reconstruct. Fowler's Solution.
	Upper neurones affected.	Exaggerated reflexes. Spastic muscles.	
	Later: Process spreads and descending degeneration involves lower neurones.	General paralysis. Contracted visual field. Optic atrophy. Ataxia. Diminished reflexes. Flaccid muscles. Atrophy.	
	Bulbar centres become involved.	Death from paralysis of deglutition or respiration.	Opium if necessary to avoid distress.

LATERAL SCLEROSIS.

Etiology.	*Pathology.*	*Symptoms.*	*Treatment.*
Infection. Traumatism. Syphilis.	Gradual primary sclerotic degeneration of pyramidal (motor) tracts.	Gradual onset. Heaviness of limbs. Stiffness of muscles. Gradual motor paralysis (beginning in legs).	Alterative and reconstructive: Ergotin to tone up capillaries. Chrom. Sulph. to check hyperplasia. Hyoscyamus and Gelsemium as antispasmodics. Nuclein to nourish nerve-tissues. Frequent warm baths.
Myotrophic Form: Attacks central part of pyramidal tracts.	Upper neurones only involved.	Exaggerated reflexes. Ankle clonus. Spastic muscles (spastic gait). No R. of D. No atrophy.	Galvanism and Faradism to affected muscles.
Amyotrophic Form: Attacks motor roots and peripheral motor neurones.	Upper and lower neurones involved.	Diminished reflexes. Flaccid muscles (dragging gait). R. of D. Atrophy.	Galvanism and Faradism to affected muscles.
	Process usually self-limited but no restoration occurs.	Symptoms usually come to a standstill.	

LOCOMOTOR ATAXIA (TABES DORSALIS).

Etiology.	*Pathology.*	*Symptoms.*	*Treatment.*
Syphilis. Exposure and traumatism.	Slow degeneration of sensory roots and tracts.	Onset gradual. Lightning pains. Visceral crises (Heads' law).	Anti-syphilitic. Baths and salicylates for the pains.
Usually attacks entire sensory tracts of cord, including sensory roots, but is sometimes limited to cervical, dorsal or lumbar segments.	Lower sensory neurones involved but not motor.	Reflexes diminished. No atrophy but optic. No motor paralysis. R. of D. present. Muscles flaccid. Delayed sensation.	Alterative and reconstructive: Ergotin to tone up capillaries. Chrom. Sulph. to check hyperplasia. Hyoscyamus and Gelsemium as antispasmodics. Nuclein to nourish nerve-tissues. Frequent warm baths.
	Meinert's fibres involved.	Argyle-Robertson pupil.	
	Sensory paths impaired.	Ataxia (Romberg). Anesthesia.	
	Diseased area demarked from healthy portion.	Girdle sensation (occasional).	
	Bladder and rectal involvement.	Impotence. Involuntary urination and defecation.	

SYRINGOMYELIA.

Etiology.	*Pathology.*	*Symptoms.*	*Treatment.*
Malformation of spinal column, resulting in dilatation of canal (hydromyelus). Thrombosis.	Scattered cavities in gray matter of cord.	Motor weakness. Muscular twitchings. Paradoxical sensory function reaction to touch, but not to temperature and pain. Brown-Séquard's syndrome.	Alterative and reconstructive: Ergotin to tone up capillaries. Chrom. Sulphate to check hyperplasia. Hyoscyamus and Gelsemium as antispasmodics. Nuclein to nourish nerve-tissues. Frequent warm baths.
	May be confined to central portion.	Muscular contractures (spastic). Exaggerated reflexes.	
	Or: May involve the anterior horns.	Muscular atrophy. Diminished reflexes.	
	Usually invades the bulbar neurons.	Death from paralysis of respiration or deglutition.	

PARALYSIS AGITANS.

Etiology.	*Pathology.*	*Symptoms.*	*Treatment.*
Trauma. Falls, injuries, etc. Emotional excitement. Constitutional diseases.	Sclerotic atrophy of cerebral neurons. Increase of neuroglia in posterior columns, causing excitation of reflex collaterals which arborize around the motor routes.	Muscular trembling. Nodding of head (continuous). Stiffness of muscles. Loss of "gravity."	Lukewarm baths. Massage. Gelsemium and Hyoscine, antispasmodics. Arsenic (Fowler's solution) and Ergotin as alteratives. Treatment is only palliative.

CEREBRAL GUMMA.

Etiology.	*Pathology.*	*Symptoms.*	*Treatment.*
General syphilis.	Growth of gumma in the brain tissue, which may be diffuse.	Slow full pulse. Headache, severe and persistent. Unequal pupils. Projectile vomiting. Exaggerated reflexes. Choked disc. Dull area of skull percussion. Wassermann positive.	Vigorous anti-syphilitic treatment. "606" injections. Mercurial intravenous injections, or inunctions.
	Or:		
	May be encapsulated and suddenly rupture into the brain tissue.	Sudden loss of consciousness and motor power of varying extent.	Potassium iodide in large doses. Nuclein as reconstructor.

INFANTILE PALSY.

Etiology.	*Pathology.*	*Symptoms.*	*Treatment.*
Defective intra-uterine. Development.	Porencephaly. Cystic degeneration of brain. Maldevelopment of brain.	Defective mentality (idiocy, imbecility). Paralyses: Hemiplegic. Diplegic. Monoplegic.	For intra-uterine cases: Antispasmodic and sedative. Gelsemium and Bromides.
Injuries at birth.	Meningeal hemorrhages. Brain cysts. Brain atrophies.	Convulsions. Epileptiform seizures. Spastic muscles. Contractures.	For meningeal and hemorhagic cases: Absorptive and reconstructive. Mercury, Iodoform and Phytolacca. Galvanism and Faradism.
Acquired.	Brain hemorrhage. Chronic meningitis. Hydrocephalus. Encephalitis.	Exaggerated reflexes. Athetosis. No atrophy. No R. of D.	For cystic cases: Operation to drain cysts (palliative only).
	Eventual secondary descending degeneration of neurons.	Death by implication of respiratory nerves.	For all cases the chief treatment is symptomatic and care of the person.

Etiology.	*Pathology.*	*Symptoms.*	*Treatment.*
Exposure. Constitutional disease.	Gradual degeneration of sensory and motor tracts of cord.	Slight fleeting pains. Slight muscular twitchings. Gradual anesthesia (bilateral). Gradual paralysis (bilateral).	Alterative and reconstructive: Ergotin to tone up capillaries. Chrom. Sulph. to check hyperplasia. Hyoscyamus and Gelsemium as antispasmodics. Nuclein to nourish nerve-tissues. Frequent warm baths.
Cervical, dorsal or lumbar, according to segments involved.	Diseased process demarked from healthy portion.	Girdle sensation.	
	Sensory paths impaired.	Ataxia.	
	Meinert's fibres involved.	Argyle-Robertson pupil (in cervical myelitis).	
Diffuse Form: Attacks entire section of cord.	Roots of lower neurons involved.	Reflexes diminished. Muscles flaccid. Atrophy rapid. R. of D. present. Dragging gait.	
Transverse Form: Attacks only transverse gray matter of cord.	Only transverse (upper) neurons involved.	Reflexes exaggerated. Muscles spastic. Atrophy slow. R. of D. absent. Spastic gait.	Galvanism to affected muscles. Reconstructive: Phosphorus.

CEREBRO-SPINAL SYPHILIS.

Etiology.	*Pathology.*	*Symptoms.*	*Treatment.*
Syphilitic infection.	A myelitis of syphilitic origin, attacking the gray matter of cord, and brain irregularly.	Same as myelitis, but irregular and asymmetrical, and atypical. Wassermann positive. Lumbar puncture shows palladia in spinal fluid.	Vigorous anti-syphilitic treatment. "606" injections. Mercurial intravenous injections, or inunctions. Potassium Iodide in large doses. Nuclein as reconstructor.

FACIAL PARALYSIS.

Etiology.	*Pathology.*	*Symptoms.*	*Treatment.*
Exposure to cold. Diseases of middle ear. Infections. Pressure by tumors.	Degeneration of peripheral portion of seventh nerve, involving:		Remove cause. Heat to face. Salicylates in large doses. Galvanism to facial muscles to maintain nourishment and tonicity.
	Below the Fallopian canal— Facial N., Post-auricular N.	Complete motor paralysis seen in smoothness of face. Inability to close eye, to drink, to whistle.	
	In the Fallopian canal— Facial N., Post-auricular N., Chorda Tympani N., Stapedius N.	Complete motor paralysis. Diminished saliva. Increased hearing. Impairment of taste in anterior two thirds of tongue.	
	Above the Fallopian canal— All the above, Gustatory.	All above symptoms. Complete abolishment of taste.	

OCULAR PARALYSIS.

Etiology.	*Pathology.*	*Symptoms.*	*Treatment.*
Acute infections. Injuries. Tumors. Exposure. Spinal diseases.	Degeneration of the third cranial nerve either nuclear or peripheral.	Paralysis of all extrinsic muscles of the eye, except the external rectus and superior oblique. External strabismus. Immobility of eyeball. Diplopia. Dilated pupil. Paralysis of accommodation. Ptosis of upper lid.	When peripheral: Remove cause. Heat to face. Salicylates in large doses. Galvanism to facial muscles to maintain nourishment and tonicity. When central: Treat underlying condition of which it is a part—tabes, syphilis, etc.

EPILEPSY.

Etiology.	*Pathology.*	*Symptoms.*	*Treatment.*
Neurotic heredity. Defect in bio-plasm. Lack of "check action" in fetal development. *Exciting Causes:* Developmental stresses. Trauma. Reflex irritations	Defective development. Physical overtops mental.	Gross, heavy physique. Mental debasement. Inability to learn. Inordinate appetite. Somnolence. Congestion.	Thorough elimination and disinfection of bowels. Colchicum to stimulate hepatic metabolism. Rigorous diet—quantity minimum; quality, simplest. Plenty of water and citrous drinks. Regular habits. Frequent warm baths and friction. Withdraw Sodium Chloride from food and substitute Sodium Bromide in same quantities only. Otherwise, *do not give bromides.*

Etiology.	*Pathology.*	*Symptoms.*	*Treatment.*
	Defective metabolism. Nerve tissues receive split cholin products instead of lecithin. Irritable cortex.	Tonic clonic spasms (flexor). Severe, with loss of consciousness, frothing at mouth, biting of tongue, involuntary relaxing of sphincters, etc., followed by fatigue and sleep (grand mal). *Or*: Mild motor spasms (flexor), with partial loss of conscious-power (petit mal). *Or*: Mere temporary suspension of mental integrity (psychic equivalent). Spasms often occur during sleep (nocturnal epilepsy). Intervals vary from weeks and months to minutes. (Status epilepticus.)	
	Final degeneration of brain cells.	Epileptic dementia.	

Etiology.	*Pathology.*	*Symptoms.*	*Treatment.*
Neurotic heredity. Defect in bioplasm. Excessive "check action" in fetal development. *Exciting Causes:* Developmental stresses. Trauma. Reflex irritations.	Defective development. Mental outstrips physical.	Undersized physique. Mental precocity. Easily fatigued. Anorexia. Anemia.	Complete *rest* absolutely essential of body and mind. Fresh air (see air). Generous, nutritious diet. Mild hypnotics to procure sleep (Chamomile). Bitters to increase appetite. Colchicum to assist liver metabolism. Fowler's Solution and Nuclein to combat anemia. (Child must be under firm but gentle control, and be made to lie down, whether sleeping or not, most of the time.)
	Deficient assimilation. Cerebrum anemic and irritable. Nerve centers poorly nourished. Organs of body inadequate to demands.	Extreme nervousness. Muscular twitchings. Athetosis. Jactitations. Insomnia. Dyspepsia. Endocarditis. Valvular lesions. Rheumatism. Constipation.	
	Imbalance usually adjusts itself with adolescence.	Recovery the rule after puberty.	

Etiology.	*Pathology.*	*Symptoms.*	*Treatment.*
Neurotic heredity. Defect in bioplasm. Lack of "check action" in fetal development. *Exciting Causes:* Developmental stresses. Trauma. Reflex irritations.	Psychic instability. Usually an infantile condition of the psyche.	Sensory disturbances: Anesthesia. Hyperesthesia. Hemianesthesia. Pain. Motor disturbances: Paralysis. Contractures. Vaso-motor and secretory disturbances. High temperature (106). Disturbances of Vision. Hearing. Taste. Smell. Vague neuroses. Unconsciousness. Convulsions (major hysteria). Spells of uncontrollable emotion (minor hysteria). No definite objective. Neuronic symptoms.	Moral control and suasion. Removal from sympathizing friends. Faradism. Cold water. Bathing and rub-downs. Massage. Fresh air and exercise. Arsenic as reconstructor. Hypnosis and suggestion. Suggestion and assurance. Forcible diversion, such as an emetic or cathartic. Strong faradism.

NEURASTHENIA.

Etiology.	*Pathology.*	*Symptoms.*	*Treatment.*
Neurotic heredity. Defect in bioplasm. Excessive "check action" in fetal development. *Exciting Causes:* Developmental stresses. Trauma. Reflex irritations.	Neuro-mental instability and inadequacy to demands of living. Defective metabolism.	Introspection. Hypochondriasis. Phobias. Imperative ideas. Anxiety. Insomnia. Forgetfulness, but not of essentials. Alternating elation and depression. Vertical headache. Backache. Anorexia. Dyspepsia. Easily fatigued. Palpitation. Vaso-motor disturbances. Paresthesias. Sexual impotence. All symptoms better under excitement.	Moral control and suasion. Tepid baths and rub-downs. Faradism. Plain nourishing diet. Hot milk at night for insomnia. Moderate exercise. Colchicum to stimulate hepatic metabolism. Citrous drinks. Arsenic and Lecithin as reconstructives. Suggestion and assurance. *Do not give Bromides or Strychnine.* The less drugs the better. Important thing is to pursue a steady systematic hygienic and moral course.

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In the face of purulent vaginal processes exerts the same definite, satisfying germicidal efficiency that it does in the presence of purulent conditions elsewhere.

By means of douches and tampons, the vaginal walls and uterine neck may be subjected to the continuous effect of **Ecthol**—and that means destruction of germ life.

TO FURTHER INCREASE THE VALUE OF IODINE by combining with it those vegetable alteratives whose clinical usefulness has been thoroughly demonstrated—

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Represents such a combination—is to secure the largest measure of Iodine effect from minimum dosage. This is a point worthy of the widest recognition, in view of the well-known proneness of Iodine to cause such gastric and other distress as to often necessitate its withdrawal —and this is a feature which emphasizes the need of making a careful choice of the form in which to administer this valuable drug.

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